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No. 6. Observations on Canine Encephalitis *

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The second session of the morning of Tuesday, September 14th, 1948, was spent in discussion of the paper prepared for Congress by Mr. MacIntyre, Dr. Trevan and Dr. Montgomerie. Inasmuch as this contribution, like that of Mr. Shuttleworth earlier in the series, represented a response to an emergency appeal by the Provisional Committee necessitated by a withdrawal, it was gratifying to all concerned to find that it attracted what probably ranks as a record attendance for consideration of the concluding paper of an N.V.M.A. Conference. Indeed, there was an embarrassment in such interest, for the crowded assembly in the Cambridge Hall included would-be participants whose rightful claim to be heard was denied them through shortage of time, intensified by the unfortunate attempt to dispose of two major subjects in one morning. In an endeavour to remedy matters, the opportunity has since been given to intending speakers to forward their contributions for inclusion in the discussion, and these will be found following the report of the general discussion, the authors having been enabled to deal, in their reply, with the points raised in these supplementary offerings.

On this occasion the chair was taken by Mr. D. D. Ogilvie, B.Sc., M.R.C.V.S., who, in introducing the essayists, mentioned that the paper about to be presented had been brought in to replace another which for various reasons it was found impracticable to give. Time was rather limited at this session and therefore it would have been inopportune to carry out any further formalities. He therefore straight away asked Dr. Montgomerie to introduce the paper.

INTRODUCTION

Dr. R. F. Montgomerie: I think most members appreciate that this is really a substitute paper. It was not our intention to present our work at this stage, for we realise that our study of these encephalitic conditions of the dog is far from complete. We were, however, persuaded that a paper on this subject, even in the present limited state of knowledge, would be acceptable to you and form a good basis for the discussion of a set of conditions which, particularly during the past three years, has occupied the attention of almost all who are engaged in small-animal practice. Some may feel disappointed that our classification has had to be based so largely on histological evidence. We do hope, however, that our description of what we have called "hard pad disease" is such that clinicians can recognise it as an entity and will not hesitate to discuss its features as encountered in general practice.

Special interest attaches to our failure to reproduce "hard pad disease" as it occurs in the field. Beyond doubt, we have a virus which produces a pathological lesion in the brain with symptoms of encephalitis but requires the concurrent presence of some condition to permit this. The epidemiology is, then, not without parallel in human medicine in that a similar predisposing state may account for the low incidence of serious nervous disease in some outbreaks of poliomyelitis despite evidence of a high level of infection.

The paper as published—that is, without the pathological illustrations—may only have conveyed a very sketchy picture of the grounds for differentiating the groups which we have numbered Ia, Ib, II and III. My colleague, Dr. David Trevan, who has been responsible for the histological work, will demonstrate and describe the pathology of these groups of encephalitic conditions with the aid of a series of lantern slides. Thereafter, we will enjoy your discussion of this subject and we know that the Provisional Committee made a very wise choice in having Major Pugh open it. We have stated our reasons for not referring to the published work on canine encephalitis, but it would be ungracious of us were we not to draw attention to the work of Major Pugh with the late Dr. Perdrau as his collaborator. Their observations were indeed fundamental and did much to focus attention on the fact that encephalitis in the dog occurred quite apart from the distemper syndrome.

The paper, which had been circulated to members, was as follows:

The title of this paper was chosen to afford us the opportunity to describe our findings, in an incompleted piece of work, in a field which is of particular and topical interest to those engaged in small-animal practice. It is not our intention to review and discuss the work of the many authors who have probed this difficult subject—canine encephalitis. That might be regarded as an unnecessary occupation of the time at the disposal of this meeting. It seemed better that we should give an account of our current views and the ground on which we base them, as a stimulus to others to describe their experience and air their views, to the benefit of us all.

Strictly speaking, the term encephalitis should be applied only to conditions in which there is an inflammatory change in the brain. However, it has acquired a clinical application far beyond that of the pathologist, so that to-day it is common to find veterinary practitioners describe as encephalitis any condition in which the symptoms suggest involvement of the brain. These symptoms vary widely; from a change in temperament to screaming fits, from a lack of muscular co-ordination to coma or convulsions.

Many cases showing these symptoms, particularly those of the less grave type, are not suffering from a true inflammation of the brain, and quite a number recover following simple treatment. We have seen dogs with such continuous convulsions that they could rarely stand, return to normal within a few days, following a change of diet to one containing an ample supply of meat. This may have been due to the removal of a toxic constituent in the original diet, possibly that associated with agenised flour as suggested by the observations of Sir Edward Mellanby. In other cases, toxaemia arising during the course of bacterial infection has set up symptoms which have led to a diagnosis of encephalitis. In some of the carcases sent to us by practitioners interested in obtaining material for our encephalitis investigation, the only lesion was a purulent pneumonia, yet the history was one of cerebral disturbance. Uraemia following kidney damage due to leptospira infections or due to dehydration following a sustained febrile illness or continuous vomiting and diarrhoea, is often associated with convulsions.

It is generally accepted that certain deficiencies are characterised by symptoms related to dysfunction of the central nervous system. Lack of aneurin (vitamin B_1) in the diet of the dog leads to loss of appetite, nervousness, convulsions and chorea; and in these cases the administration of large doses of the vitamin brings about dramatic recovery. Pantothenic acid is needed by the dog, and a deficiency quickly leads to gastritis, enteritis, convulsions and coma. On *post-mortem* examination the gastritis and enteritis is accompanied by intussusception. Several such cases have been among our series of clinically accepted encephalitis.

Frank cases of rickets in puppies not uncommonly show convulsions; and the convulsive states associated with helminthiasis in the young are another example of the type of condition which can be mistakenly diagnosed as encephalitis.

Many cases of so-called encephalitis are, however, associated with the invasion of the brain by an infectious agent. In some instances this agent is the true distemper virus of Laidlaw and Dunkin, yet the strains of virus isolated from these cases have had no real neurotropic properties. Injected by a variety of routes, including the intra-cerebral, these strains have produced in susceptible dogs typical distemper, without any sign of nerve or brain involvement. Ten days after the subcutaneous injection of virus from a typical case, the brain of a susceptible dog may have a

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high virus content without the dog having shown signs of nerve cell damage, other than some dullness. Of the many thousands of susceptible dogs injected during the production and testing of distemper virus for use in immunisation, less than 20 have shown signs of nervous disease. Another virus associated with cases of encephalitis, which we will describe later, has so far failed to produce that condition when injected by a variety of routes into our susceptible dogs. But, encephalitis due to one or other of these infectious agents is a common occurrence in most small-animal practices and may, indeed, occur after the use of live virus in immunisation. We are driven to the conclusion that, particularly in these days of difficulty in procuring proper food for dogs, impaired nutrition is an important factor in the causation of canine encephalitis. It may act either by preparing the way for the entry of virus which does not normally attack nervous tissue or by creating a state in which minimal damage shows to maximal extent. Some such hypothesis is necessary to explain the observations which we have made; for the only true neurotropic virus infecting the dog is, apparently, that of rabies.

We recognise that any of these factors may be responsible for the production of nervous disorder, clinically diagnosed as encephalitis, but that the cases associated with a virus infection are usually more severe and almost always more difficult to treat successfully. For a period of years we have been attempting to sort out these conditions by investigating their aetiology and pathology. To this end we have received from practitioners much material, mostly *post-mortem*, but occasionally in the late *ante-mortem* stages. It was soon apparent that a classification could not be solely based on the history and symptoms supplied or observed. This is not surprising, since the signs in any case must depend largely on the part of the central nervous system involved, rather than on the type of lesion existing. Nevertheless, one or two clinical characteristics appear to attach to a particular group in our tentative classification, which is based on the histological picture and, in part, on evidence as to aetiology. This classification is as follows:

Group I.—In which a filterable agent, infectious to the ferret, can be demonstrated in brain and other tissues, is divided into two sub-groups:—

Group Ia.—Associated with infection with the true distemper virus of Laidlaw and Dunkin; the brain lesion is essentially that of destruction of nerve cells without inflammatory changes or demyelination.

Group Ib.—Associated with infection with a virus differing from that of distemper; the brain lesion is a demyelinating and inflammatory encephalitis. A remarkable hyperkeratosis of the foot pads is so common a feature of these cases that the condition has come to be known as "hard pad disease."

Group II.—A demyelinating, inflammatory encephalitis which is not infectious to the ferret, but which in one of several attempts was transmitted to a dog.

Group III.—An encephalitis, inflammatory but not demyelinating, which is apparently not infectious.

Group IV.—An encephalitis apparently caused by protozoal invasion of the brain; a toxoplasmosis, probably rare in occurrence.

Group V.—Miscellaneous conditions, the majority of nutritional or toxic origin.

We have every reason to believe that the conditions which we have placed in the first four groups are entities. We will describe their main features, particularly those of Group Ib ("hard pad disease") since we consider it responsible for the epizootic which has caused so much concern during the past two or three years. We will later illustrate our findings by a series of lantern slides and we have a set of specimens available for inspection.

Group Ia.

This encephalitis may occur towards the end of the acute phase of typical distemper, generally following pneumonia, but is more common some weeks after apparent recovery. Its appearance while symptoms of distemper are still in evidence is almost confined to cases in young puppies, and the nervous symptoms are largely due to a secondary toxaemia or septicaemia. The rigors, stupor and coma often suggest encephalitis. In the dog recovered from the acute phase of distemper, the symptoms are of the choreic type, appearing first in the masseter and temporal muscles, and spreading to the limbs and body. Epileptiform convulsions, while they do occur, are not prominent in the early stages, and are seldom the only nervous symptom.

Post-mortem examination shows little that would suggest encephalitis. The brain may be congested; a few tiny haemorrhages and an excess of cerebrospinal fluid may be present. A leptomeningitis may be suspected, but more usually the brain is normal in appearance. Other organs are generally congested, the lungs showing collapse and pneumonia, the lymph glands enlargement and the bowel wall enteritis, but the whole picture may be complicated by the effects of secondary infections.

Histologically, the brain shows severe destruction of nerve cells, particularly in the cortex, and many small haemorrhages. Some gliosis may be present, but there are no other inflammatory changes. We have seen no inclusion bodies, and there is no demyelination. In the lungs there is a histiocytic interstitial pneumonia, in small patches, which nearly always becomes secondarily infected and infiltrated with polymorphs. The bronchial epithelium contains cytoplasmic inclusion bodies.

The injection of emulsions of brain and other tissue from cases of this sub-group has regularly produced typical distemper in the ferret. After an incubation period of nine to eleven days, at the most 15 days, dullness, watery ocular and nasal discharge, swelling of the eyelids and congestion of the muzzle appear. The discharges soon become purulent and vesicles form in the skin of the lips. The eyelids become glued together, the vesicles rupture and scabs form. Recovery is rare and death usually supervenes within three days of the appearance of symptoms. Nervous symptoms, other than a hyperaesthesia, are rarely seen, probably because the duration of the disease in the ferret is so short. Laidlaw and Dunkin did see one or two cases in which nervous symptoms were prominent, and sections of the brain from these cases showed only death of nerve cells.

Prior to 1945 all our encephalitis cases from which a virus was demonstrated in this way had the histological picture described above.

The strains of virus from these cases produce typical distemper in the dog, without the appearance of nervous symptoms, and they are readily neutralised by anti-distemper serum.

Group Ib.

Reports that distemper prophylactics had failed to protect and that immunised dogs became affected with so-called nervous distemper, first focused our attention on this disease. Instances in which repeated doses of serum had failed to protect in-contacts, and the use of as much as 400 c.c. of serum at the very outset of illness had afforded no benefit, were brought to our notice. Yet the serum was known to be of high anti-viral content when tested against Laidlaw-Dunkin virus. The case history of many of these dogs was not typical of distemper, and ferrets inoculated with tissue emulsions from dogs that had died of this disease were unaffected after the normal 15 days' observation. It had always been our practice to test the immunity of ferrets surviving following an injection for diagnostic purposes by injecting stock distemper virus 15 to 17 days after the original injection. Although most of these ferrets developed distemper in the standard period of eight days after the challenge dose, some were ill with a distemper-like disease within four or five days. Such illness could only be related to the original injection, and when the period of observation was extended to one month, it was seen that in some cases a disease appeared in the ferrets long after the 15-day interval. We then realised that some of the former tests for virus must have been mistakenly accepted as negative.

In the autumn of 1946 Dr. Margret Scheitlin of Zurich visited our laboratories, and during our discussion of canine encephalitis, described a hardening of the foot pads which she had seen in Switzerland. Where this remarkable lesion was observed she could predict with fair certainty that nervous symptoms would develop within a few days. We were soon able to confirm this observation in our own cases, and practitioners to whom we mentioned this curious circumstance have found and presented us with many examples of it. The condition which we classify as Group Ib has this symptom as its most outstanding and characteristic feature, and has come to be known as "hard pad disease."

This disease affects dogs of all ages. We have seen it in puppies that sickened when six days old, and have recently had a case in a nine-year-old dog.

When catarrh is present, the discharge is thin and watery and rarely of the copious purulent type so common in distemper. Many acute cases show little or no nasal discharge, but in those of some standing a rather scanty secretion crusts at the nostrils.

A moderate tonsillitis with enlargement of the cervical glands and some swelling of the other lymph glands is a regular feature.

Usually there is fever, irregular and intermittent; sometimes the temperature is as high as 106·5° in the early stage. Yet, in some cases a sub-normal temperature persists through the illness. The febrile reaction is a contrast to the diphasic fever so characteristic of distemper.

Severe diarrhoea is common. In fact, violent diarrhoea and, in greyhounds, dysentery are almost constant symptoms.

Cases often show fast, shallow breathing suggestive of pneumonia, but no signs of consolidation can be detected on clinical examination. Indeed, *post-mortem* examination of these cases reveals no more than a few small patches of interstitial pneumonia, seldom

extensive or secondarily infected, although pulmonary oedema and congestion are common. There is evidence of acute degeneration of the heart muscle fibres, but this is hardly sufficient to account for the pulmonary symptoms on a basis of heart failure, and it may be that the oedema of the lungs is neurogenic in origin.

The outstanding and relatively constant feature of this disease is the remarkable hardening of the foot pads. We believe that it occurs to some degree in every case but varies from a hyperkeratosis only recognisable histologically to very marked thickening and hardening which results in distinct tapping as the dog walks on a firm surface. Grossly thickened pads may have a keratin layer a quarter of an inch thick. Others remain thin, but have the hardness of metal, and occasionally a "corn" is present in them. The pads are seldom painful but some feel hot. The hardening becomes so marked that when pressed from the side they are unyielding, in contrast to the rubbery nature of the normal pad. This feature appears within one week of the onset of illness, the pads at first showing little more than slight swelling, and some disappearance of the papillae, which gives a smooth glistening appearance. The rate of development varies, but generally by the end of a further week the pads have the characteristic appearance. In some cases which have been affected for several weeks, a similar hyperkeratosis of the nose has been seen.

In the fulminating type, fits are often the first symptom observed, and after a succession of severe convulsions the dog may be dead within one week. In the majority of cases the symptoms of encephalitis appear at the same time as, or within a few days of, the hardening of the pads. Occasional convulsions with loss of consciousness, lasting only a few minutes, soon become more frequent and more lasting until the animal becomes comatose and dies. Screaming fits, blindness and paraplegia are less common; chorea, where it does occur, is chiefly seen in the more chronic cases.

Cases have been reported in which the hardened pads desquamated, but in about 90 per cent. of dogs affected, encephalitis leading to death (or destruction) has followed within two weeks of the appearance of the pad condition.

The *post-mortem* picture is somewhat variable depending on the severity of the disease. In some, little is found beyond the changes in the foot pads, an enlargement of the lymphatic glands and some congestion of the lungs. In many, a notable feature is congestion, haemorrhage and oedema of the lungs, with small scattered patches of grey consolidation, usually sub-pleural, and not extending more than a few millimetres into the lung substance. These patches are more easily felt than seen. Excess of clear pleural fluid, excess of pericardial fluid and sub-epicardial haemorrhages are common. Gastritis is usually marked, and sub-peritoneal haemorrhages occur along the lesser curvature of the stomach. Severe enteritis throughout the duodenum, and elsewhere patchy, is usual, and the contents of the bowel are commonly blood stained. The spleen is markedly congested, the liver similarly affected, and the gall bladder engorged. A haemorrhagic cystitis is common. The inflammatory condition of the brain is not evident to the naked eye, apart from the appearance of meningeal congestion.

Histological examination, besides confirming these *post-mortem* findings, shows that the brain lesion is essentially a demyelinating encephalitis occurring in small scattered focal areas, typically in the cerebellar peduncles and folia; often, when elsewhere, submeningeal or sub-ependymal. There is glial proliferation, swelling and proliferation of the vascular endothelium sometimes with histiocytic "cuffing" of vessels. Intranuclear inclusion bodies, weakly acidophil, occur in glia cells, histiocytes, ependyma cells and occasionally in meningeal endothelial cells. They do not appear in vascular endothelium as described by Green for fox encephalitis. Between the nuclei, among the glial network, can sometimes be seen structures like inclusion bodies which are presumably in the unstained cytoplasm of glia cells. There are also curious masses of finely vacuolated "colloid"-like material containing no nuclear structures but surrounded by glia-like nuclei often containing inclusions. Despite prolonged search we have never seen inclusions in nerve cells.

Lung tissue shows a picture somewhat similar to that seen in true distemper. There is a histiocytic interstitial-cum-alveolar pneumonia often in tiny patches with cytoplasmic acidophil inclusion bodies in alveolar and bronchial epithelial cells. Extensive polymorph infiltration and purulent resolution are much less common than in distemper. There is severe congestion, haemorrhage and oedema.

The pads show great thickening of the horny layer and proliferation, distortion and premature keratinisation of the prickle cells, so that deep into the horny layer are often found pyknotic nuclei and nuclear fragments from cells which have keratinised before the normal loss of their nuclei.

The injection into the ferret of an emulsion of foot pad, of brain, of lung or of spleen tissue from these cases sets up a disease which bears some resemblance to distemper in this species. The incubation period is only occasionally less than 17 days and may be as long as 32 days, the average for a large number of observations being 23 days. In investigating cases of this group, it has been our practice to inoculate into two ferrets each of the tissue emulsions just mentioned. The variability of the incubation period has been noticeable. One ferret of the pair may show symptoms as early as the tenth day; its companion not until the twenty-eighth. Those on pad emulsion may have an incubation period of 20 days, while those infected with brain emulsion show no symptoms until the thirtieth day. This rather suggests that the ferret is an unnatural host, and that the virus finds some difficulty in establishing itself. Ferrets injected with tissue emulsion from puppies which sickened when six days old and died at ten days, showed no symptoms until after the twentieth day. The nasal discharge in infected ferrets is slight and watery, and conjunctivitis is not often purulent and rarely glues the eyelids together as in distemper. Vesicles are often absent and the muzzle is invariably moist. Although the ferret is obviously infected, the symptoms develop slowly and the disease is not violent; death may not occur until the tenth day of illness. In contrast to ferret infection with Laidlaw-Dunkin virus, recovery is not uncommon. Concurrently with the appearance of other symptoms most ferrets develop hard pads, no matter what tissue emulsion has been injected. Despite the longer course of the disease definite nervous symptoms have not been seen in ferrets and there has been no histological evidence of inflammation of the brain.

We have earlier noted that the injection of Laidlaw-Dunkin virus, no matter the route selected, produced distemper with all its classical symptoms but rarely, or never, evidence of nerve involvement. In contrast, the injection of the virus of "hard pad disease" into our experimental dogs produced only a febrile reaction, usually intermittent and lasting ten to 14 days. Invariably these dogs return to normal without exhibiting any other symptoms. Recovery after the injection of either of these viruses left the animal immune to the homologous virus. Dogs which had been proved immune to "hard pad" virus showed the typical diphasic temperature reaction when injected with Laidlaw-Dunkin virus and, conversely, distemper immune dogs often reacted to the injection of "hard pad" virus. It was noticed, however, that the reaction to "hard pad" virus was generally less severe in these cases. The injection of large doses of anti-distemper serum from several batches, some prepared as long ago as 1937, has failed to prevent infection with "hard pad" virus, and this virus has not been neutralised *in vitro* by high quality distemper serum. A proportion of dogs of unknown history, proved immune to distemper, have failed to react to the injection of "hard pad" virus. This may well have been due to their having an acquired immunity to the virus, and it suggests that non-clinical disease is not uncommon. We believe the serum protection tests and serum neutralisation tests give a more certain differentiation than immunity tests in dogs. We do not regard the two viruses as being entirely unrelated but the differences we have noted from both the immunological and clinical aspects seem quite sufficient for their acceptance as entities.

Our failure to reproduce clinical "hard pad disease" has led us to conclude that factors in addition to the virus infection are responsible for its occurrence in the field. Among other things, our attention was attracted to the frequency with which cases went on to show the full clinical condition following treatment with sulphonamides in the early phase of the disease. We have therefore observed the effect of dosage with these drugs in dogs injected with the virus. Groups of four dogs were injected with virus. One dog in each group was left as a control, the other three being given full oral doses of a sulphonamide for the first eight days after infection. A control group of dogs were given drug alone without virus. In the case of two of the drugs used, five out of the six dogs had fits from about the eleventh day onwards; that is after the drug had been discontinued. There was histological evidence of a demyelinating encephalitis in one case and of hyperkeratosis of the pads in three. None of the dogs on virus alone or on drugs alone showed any nervous symptoms. This observation tends to confirm the suspicion which arose in field work that the administration of some sulphonamide drugs is one of the factors leading to full development of the disease. At the moment we are investigating other factors which may have a similar relationship to "hard pad disease."

Group II

This disseminated demyelinating encephalitis has a histology somewhat similar to that of Group Ib but is chiefly distinguished by the failure to infect ferrets with Seitz filtrates of tissue emulsions.

Clinically, the disease is insidious in onset and slow to progress. Dogs of all ages have been found affected. Signs of generalised infection are slight or absent; there may be a mild catarrhal

conjunctivitis and a dry cough with slight enlargement of lymph glands. In many cases these signs have cleared up before the appearance of nervous symptoms, and most cases have presented as a purely nervous disease. The dogs have convulsions. They may show paralysis, chorea or ataxia. A feature which we have come to regard as characteristic is a tendency to walk in circles to one side or the other, bumping into objects but continuing the same circling movement when the obstruction is removed. General body condition is fairly well maintained, the animal usually dying in coma. At *post-mortem* examination, the brain appears normal but may be somewhat congested. The lungs are often normal but where the terminal coma has been protracted there may be a severe pulmonary congestion and oedema. There is no pneumonia, and there are no constant changes to be seen in other organs. Histological examination of the brain shows a demyelinating encephalitis; there are small foci of demyelination, gliosis, histiocytic cuffing of blood-vessels, and thickening of vessel walls. There are intranuclear inclusion bodies in glia cells near the cuffed vessels; but there is hardly any damage to nerve cells, and we have not seen ependymal inclusion bodies. The lung may show congestion and oedema, or it may appear normal; there is no pneumonia, nor are there inclusion bodies in bronchial epithelium. There are no significant changes in other organs, and the pads are normal. Ferrets injected with Seitz-filtered material from these cases remain normal. Attempts have been made to reproduce the disease in dogs in this way. Of several dogs injected intracerebrally, one developed circle walking, rigidity, difficulty in accommodation and convulsions, without loss of consciousness. Histological examination of this case showed a typical group II encephalitis; the pads were normal.

It therefore seems that there is a demyelinating encephalitis of dogs other than "hard pad disease."

Group III

This condition was fairly common ten or 15 years ago, but at present it appears to be relatively rare. Most of our cases have been in older dogs. The history is of the insidious onset of a purely nervous condition; there are no signs of respiratory disease or of generalised infection. The dog becomes dull or shows other changes in temperament; it wanders about aimlessly and has repeated fits. There is usually no evidence of permanent damage to nervous functions such as would be shown by paralysis, ataxia or sensory loss. The general condition remains good, and the dog may live for a long time. In some suspected cases, the fits have stopped and the dog has apparently recovered. In those cases that come to *post-mortem* examination there are no typical naked-eye abnormalities. Sections of the brain, however, show the most noticeable feature to be a cuffing of the blood vessels with small cells, mostly lymphocytes, lying in the perivascular space. There is no thickening of the vessel wall, and there are no inclusions, nor is there any demyelination, although where a vessel in white matter is heavily cuffed, an appearance of demyelination may be produced, as the fibres are pushed aside by the large cuff. There appears to be no damage to nerve cells. We have seen no significant change in the other organs. Ferrets injected with Seitz-filtered material have remained normal, and the few attempts that we have made to transmit the disease to dogs have failed.

Group IV

We have reserved this group for cases of protozoal infection of the brain which are known to occur in other countries, although we have only one definite case in our series. It was a case of toxoplasmosis and probably the first reference to the condition in the dog in this country.

This particular dog from a country district was reported to have been vaguely off colour and running a high temperature for two weeks before fits and temperamental changes appeared. It was destroyed and sections of its brain and other organs were submitted to us. The brain showed a large area of necrosis in the medulla; the area was sparsely infiltrated with glia and plasma cells. Many toxoplasma were present, singly and in clusters or "pseudocysts." The blood vessels in the area were cuffed with plasma cells. The lungs showed an alveolar pneumonia but no parasites could be seen in the sections.

Previous to this we had a case, a dog which showed nervous symptoms, in which the histological findings in the brain were indefinite but the heart muscle showed a destructive inflammatory lesion associated with protozoa resembling toxoplasma in clusters within the heart muscle fibres.

Group V

For the present, we have left collected together those conditions with which we have very little experience, or which appear to be associated with nutritional deficiency, intoxication or helminth infestation. Histologically they do not fit into any of the above groups.

Treatment

Some might regard this contribution as incomplete, if we did not say a word about the treatment of these conditions.

Since we have been unable regularly to reproduce the various types of disease which we have described, we have had no opportunity to study methods of treatment in controlled conditions. We have, however, collected much information from practitioners who have adopted procedures which we have suggested.

Where damage to the nervous system has been severe and complete, functional recovery never occurs, so that the dog is left with a residual symptom of the disease, such as altered mentality, defective vision, deafness or muscular incompetence, etc. Specific anti-sera have been used in the group I conditions. Anti-distemper serum has little therapeutic value in cases of distemper already showing nervous symptoms. Its value as a prophylactic is undoubtedly high. We have prepared experimental batches of serum by hyperimmunising dogs with the virus of "hard pad disease." Used therapeutically, this serum has given some encouraging results; but only where it has been used in the early phases and repeated about every five days, for a period of four to five weeks. In some cases its intrathecal injection in 10 c.c. amounts into dogs already showing nervous symptoms has been followed by definite improvement in the clinical condition. Animals in contact with this disease appear to have been protected by the injection of serum. As yet, we have been unable to produce the serum in quantities large enough for general issue. However, one cannot rely on specific treatment with confidence. The measures adopted should, then, be those directed towards assisting the natural recuperative processes. This is specially the case in "hard pad disease" in which almost all the organs become involved.

In all encephalitis-like cases early consideration should be given to diet. It must be adequate as to protein, vitamin and mineral content. The feeding of flesh should be supplemented by access to offal, yeast or other vitamin rich foods. Flour, in any shape or form should be avoided, as very little of the bread, brown or white, available in Britain to-day, is unagenised. Crude cane sugar molasses has been found of value, probably because of its content of the vitamin pyridoxine, a deficiency of which may lead to convulsions and chorea in addition to pneumonia. Molasses feeding may, however, aggravate an existing diarrhoea. Aneurin in very large doses has been used with variable effect. Certain Dutch investigators have advised the use of strong liver extract, injected intramuscularly in 2 c.c. doses on two successive days. Although this line of treatment appears to have little effect in many cases, it has been used with apparent success in some cases of chorea.

In most cases, particularly those of group I, dehydration is marked, especially when diarrhoea is prominent, and the subcutaneous or intravenous injection of glucose saline is essential. In these cases the administration of vitamin B by injection is of value because the alteration of the intestinal flora prevents synthesis of this vitamin and there is defective absorption.

We have referred earlier to the grounds on which we believe sulphonamides are contra-indicated, particularly in cases of group Ib. Where there is fever and some tonsillitis, the use of penicillin is preferable.

The pulmonary oedema in cases of group Ib is of special concern. Professor Cameron has recently observed that acute oedema of the lungs can be produced in rabbits, by injection of fibrin solution into the cisterna magna, and that this oedema can be prevented by section of the vagi or by the injection of atropine. It is possible that the oedema of the lungs seen in these dog cases is neurogenic, so that treatment with atropine would be appropriate. We have, as yet, had little opportunity to apply this suggestion.

With a view to encouraging the full intake of food and water the use of hypnotics is contra-indicated. Although they may be demanded to control screaming fits, it is advisable to try the effect of less powerful remedies such as aspirin to allay pain.

In general, treatment of these conditions is so difficult, that we would lay emphasis rather on the need for finding and applying adequate prophylaxis.

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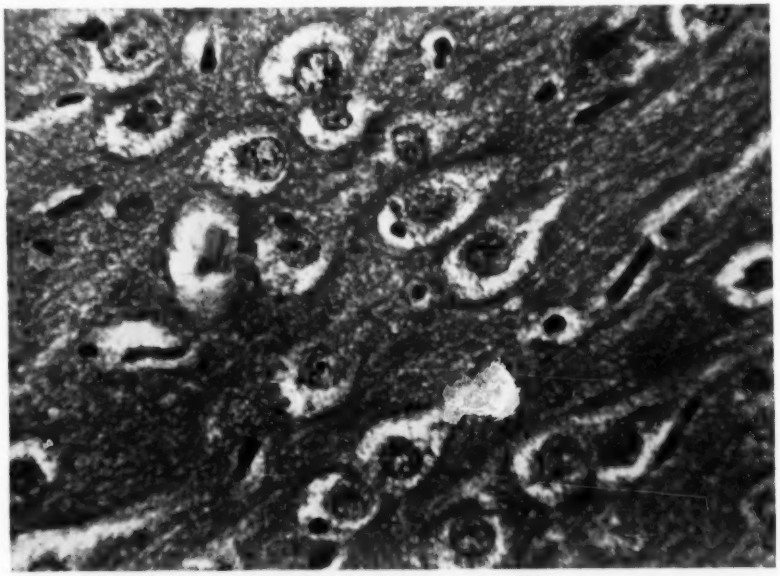
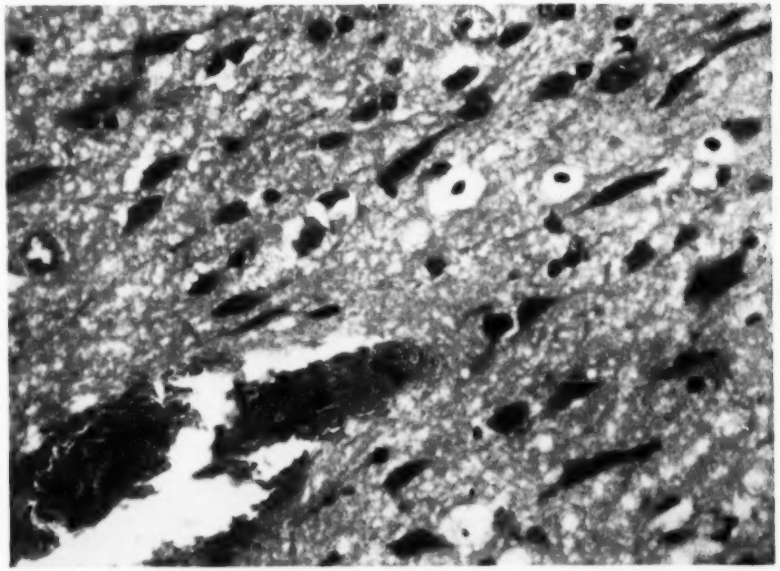
The demonstration of lantern slides and further description of the pathology of these groups of encephalitic conditions, which follow this paper, will serve to clarify the picture which we have tried to draw. There is also a small collection of pathological material, specimens and sections, which illustrate them further.

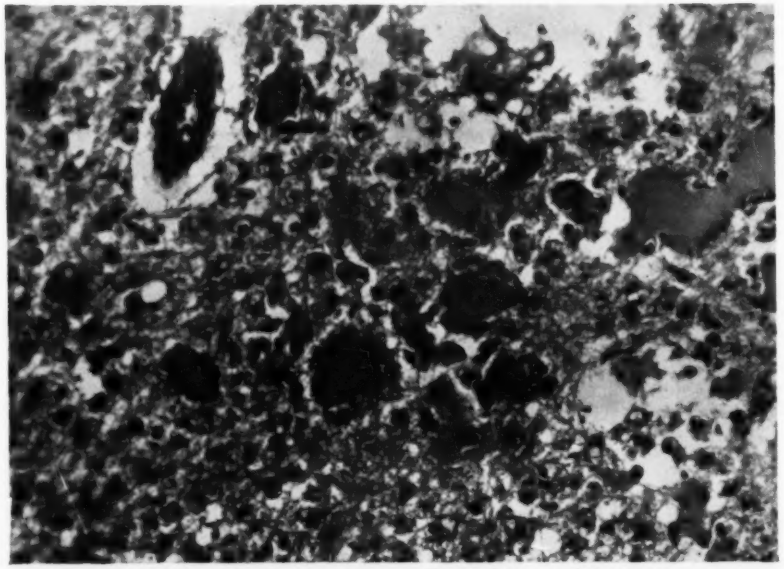
Our clinical differentiation is not easy; we look to the practitioners present to add to our knowledge in this direction. Our work is far from complete but what we have accomplished could only have been done by our having the co-operation of many practitioners to whom we look for further assistance. Our objective, now that we



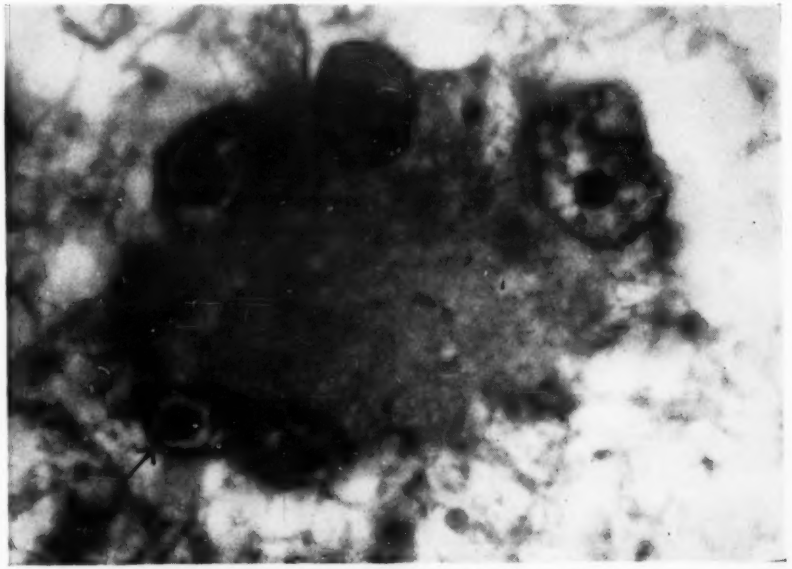
1. Normal cerebral cortex. ×480.



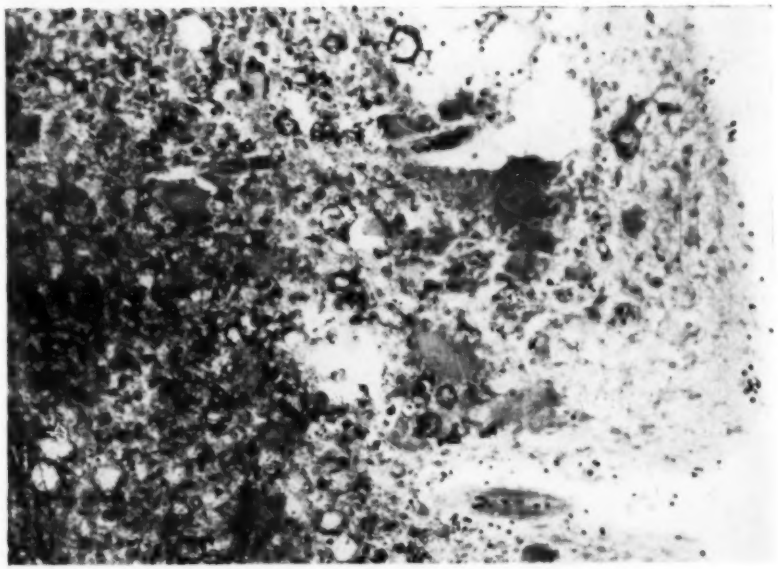
2. Distemper (Group Iᴀ), cerebral cortex. ×480.



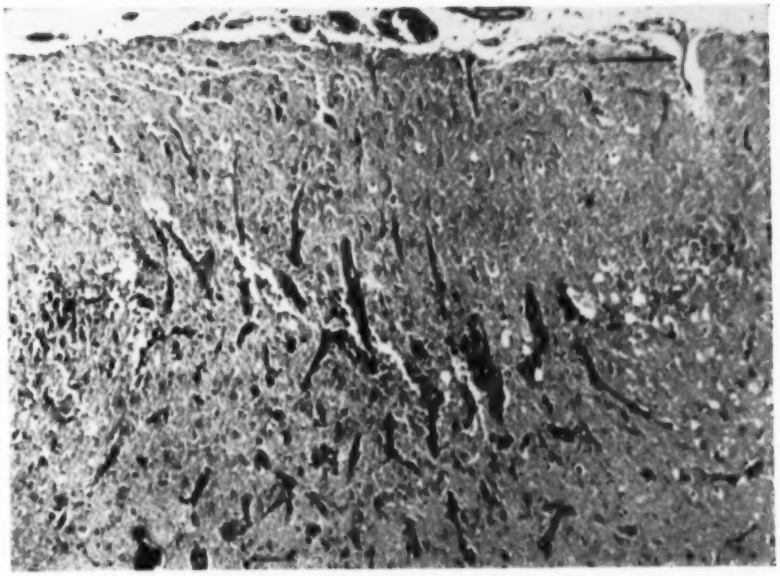
3. Medulla, hard pad disease (Group Iʙ). ×290.



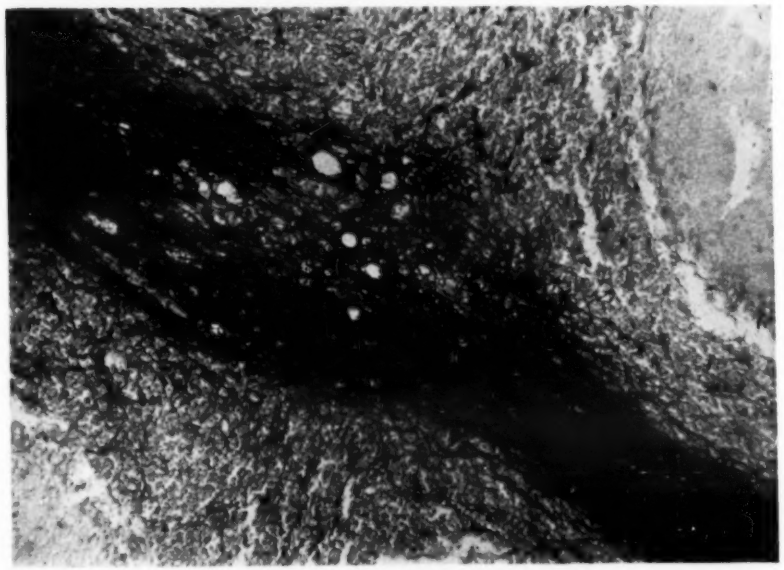
4. Group Iʙ, intranuclear "inclusions." ×2,400.



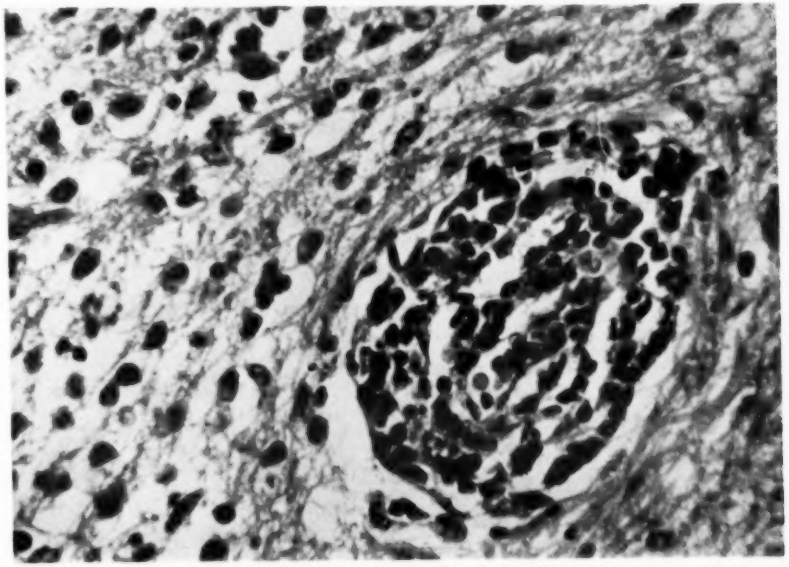
5. Group Ib, demyelination. ×170.



6. Group Ib, thickened vessels in cortex. ×68.



7. Group Ib, demyelinated cerebellar folium. ×100.

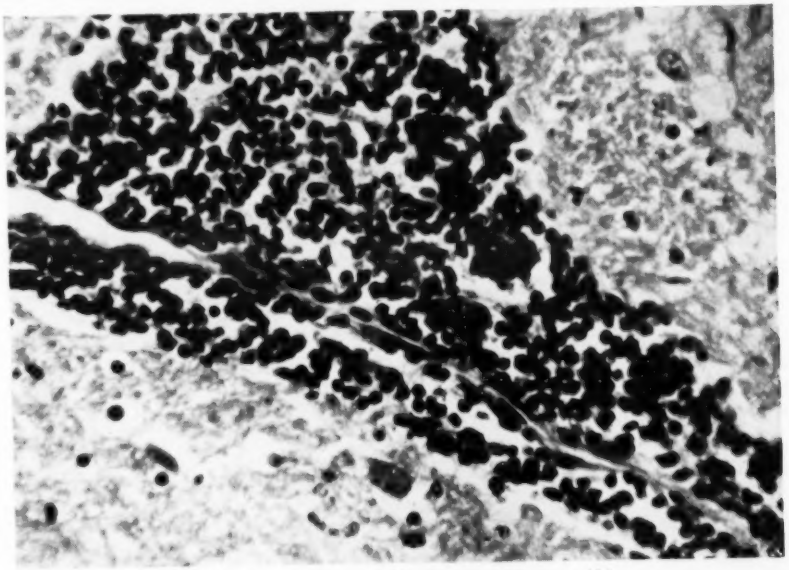


8. Group II, histiocytic "cuff." ×500.

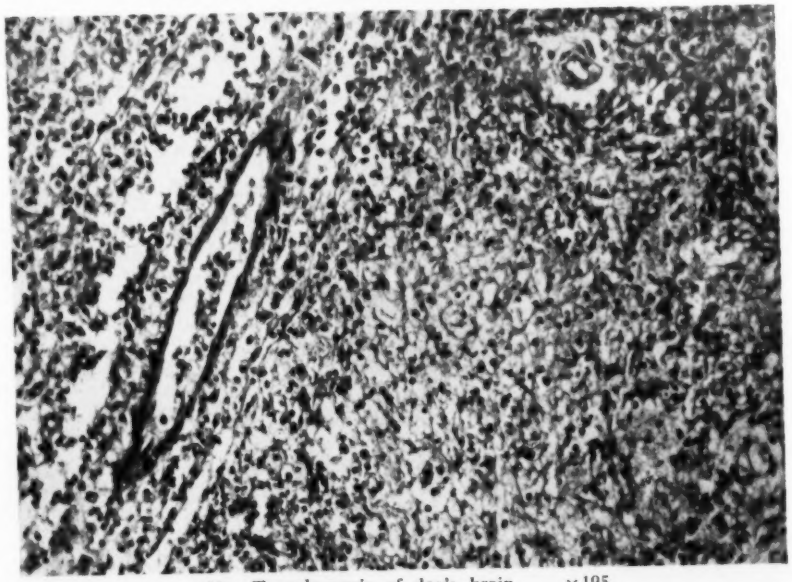
7. Group Ia, demyelinated cerebellar folium. ×100.

8. Group II, histiocytic "cuff." ×500.

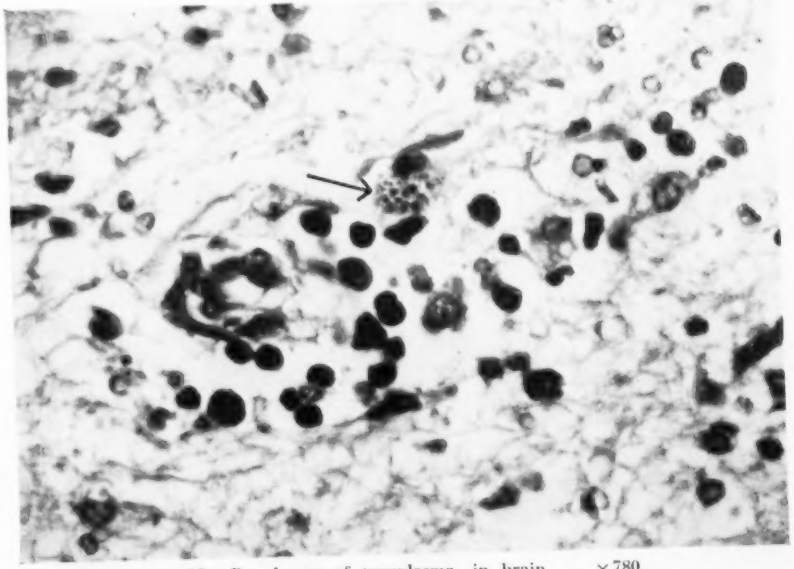
9. Group II, intranuclear "inclusions." ×4,400.



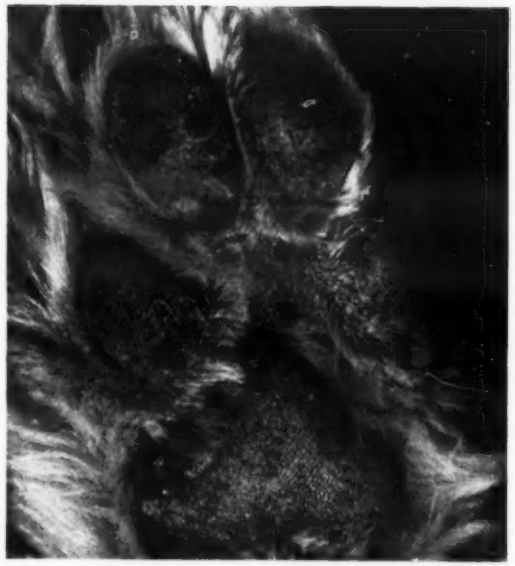
10. Group III, lymphocytic "cuff." ×525.



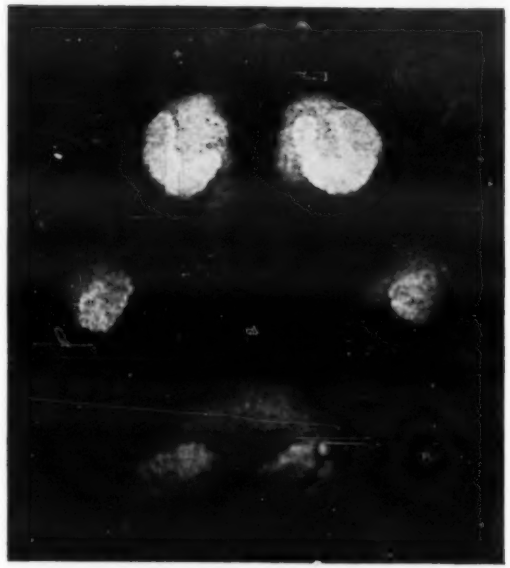
11. Toxoplasmosis of dog's brain. ×195.



12. Pseudocyst of toxoplasma, in brain. ×780.



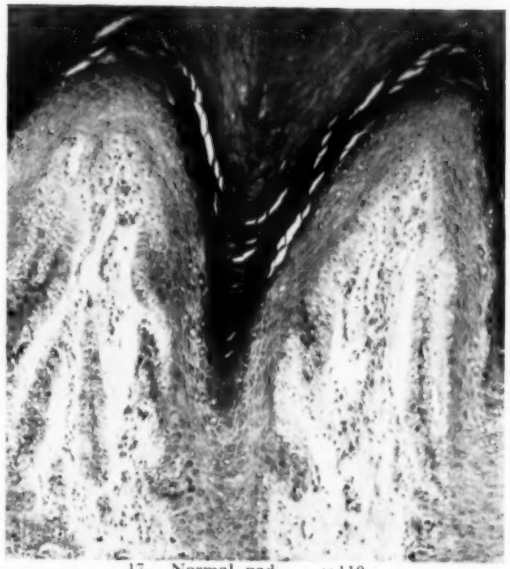
13. Early hyperkeratosis, Group Ib.



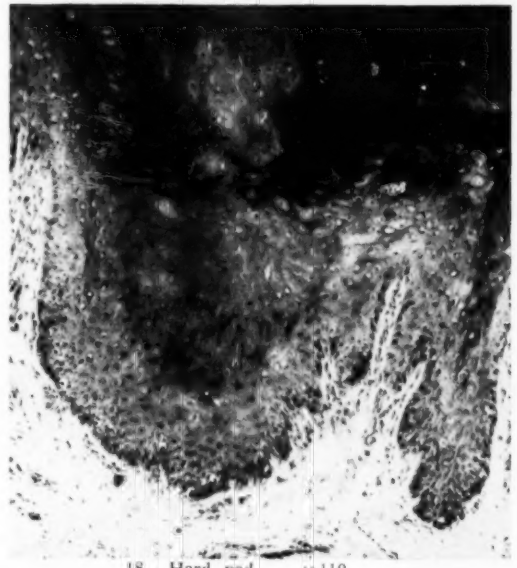
14. "Horse-shoe" corn. Group Ib.

15. Advanced hyperkeratosis, Group Ib.

16. Hyperkeratosis in puppy, Group Ib.



17. Normal pad. ×110.



18. Hard pad. ×110.

have so far sorted out these diseases, is to carry our work forward towards determining the influences which lead to their appearance, and to meeting the need for sound prophylactic and therapeutic procedures.

. . . . . .

After Dr. Montgomerie's opening remarks, Dr. D. J. Trevan discussed, with the aid of lantern slides, the histology of the various types of encephalitis as described in the paper. Some of these pictures are reproduced as illustrations to this paper.

Description of Illustrations.

1. Dog, normal cerebral cortex, nerve cells. Haematoxylin and eosin. ×480.

2. Dog, cerebral cortex, distemper (Group IA). Note shrivelled nerve cells and haemorrhage, bottom left. Haematoxylin and eosin. ×480.

3. Dog, part of area of inflammation and demyelination in upper medulla section—hard pad disease (Group IB). Note masses of "pseudocolloid" and blood vessel with thickened wall, top left. Haematoxylin and eosin. ×290.

4. Higher power view of (3) above; arrow indicates intranuclear "inclusion" body. Haematoxylin and eosin. ×2,400.

5. Same area as (3) above (lower magnification) showing demyelination. Weil's stain. ×170.

6. Group IB, patch of thickened vessels in cerebral cortex, near surface. Haematoxylin and eosin. ×68.

7. Group IB, patch of demyelination in a cerebellar folium. Weil's stain. ×100.

8. Group II, histiocytic "cuff" and histiocytic and glial infiltration, in a demyelinated area of mid brain. Haematoxylin and eosin. ×500.

9. Group II, intranuclear "inclusion." Haematoxylin and eosin. ×4,400.

10. Group III, lymphocytic "cuffing" of a vessel; wall of vessel not thickened. Haematoxylin and eosin. ×525.

11. Toxoplasma infection; part of area of softening and inflammatory infiltration of brain of dog. Haematoxylin and eosin. ×195.

12. High-power view from (11) above showing parasites in pseudocyst, near vessel which is loosely "cuffed" with plasma cells. Haematoxylin and eosin. ×780.

13. Afghan hound, early hyperkeratosis (Group IB).

14. Miniature poodle, "horseshoe corns," one of the types of moderately advanced hyperkeratosis in Group IB.

15. Terrier, advanced hyperkeratosis (Group IB).

16. Hyperkeratosis in a puppy (Group IB).

17. Section of normal dog's pad. Haematoxylin and eosin, green filter. ×110.

18. Section of hard pad (Group IB). cf. (17) and note (i) loss of sharp division between epithelium (below) and keratin (above); (ii) irregular keratinisation of epithelium; (iii) distortion and vacuolation of many of the prickle cells, which are greatly increased in numbers. Haematoxylin and eosin, green filter. ×110.

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DISCUSSION

Mr. L. P. Pugh, B.SC., F.R.C.V.S. (Sevenoaks), who opened the discussion, said: Having received the honour of opening the discussion on this paper, I should like on your behalf to congratulate the writers, firstly on the excellent presentation of a difficult subject, and secondly on their being able to incorporate some of their recent and important findings on this subject—findings which I frankly believe will constitute one of the real landmarks in the elucidation of the canine distemper problem as met with in the field. For many years prior to the publication of Laidlaw and Dunkin's work in 1925, alleged outbreaks of so-called distemper occurred in varying forms—the pneumonic, the gastro-enteric, the nervous, etc., and the severity and variation of the differing clinical pictures were attributed mostly to the action of secondary invaders. That outbreaks should at one time be predominantly pneumonic, whilst at others predominantly nervous was explained by some as being due to the distemper virus having epitheliotropic characters at one time, whilst at others neurotropic tendencies. But whatever we thought the explanation to be, we all felt confident after Laidlaw and Dunkin had devised a means of immunising against the causal virus, that the varying forms of distemper would cease to plague us if we carried out carefully their prophylactic immunisations. Unfortunately, as time went on, so-called "breakdowns" to these inoculations occurred which we attributed varyingly to faulty technique or defective material, or to a flare-up of a latent infection, etc. The one thing, however, that did emerge from all these disappointments was the fact that it was nervous phenomena which almost invariably dominated the clinical picture in these "breakdowns." I think that this fact alone began to sow the seeds of doubt in our minds as to whether there were not some other factors or infections at work in the field besides the Laidlaw and Dunkin virus. Again, ferrets placed in contact with some of these cases failed to develop distemper. The doubt was further increased by demonstration of the fact that the histological picture of brain tissue from these "breakdowns" was completely different from that clearly described by Laidlaw and Dunkin. Further, experience showed that even the histological changes in these encephalitis cases were markedly different at times. MacIntyre, Trevan and Montgomerie in these circumstances and on the strength of a great deal of work on the subject have put forward for our consideration the following tentative classification of these different forms of canine encephalitis as occurring *in this country*, and have divided the same into five main groups.

Group I.—This group, in which a filtrable agent, infectious to the ferret can be demonstrated in brain tissue, is divided into

IA Laidlaw and Dunkin virus: brain lesion essentially nerve cell destruction without inflammatory changes or demyelination;

IB A virus differing from L. and D. virus. Brain lesion essentially a demyelinating and inflammatory encephalitis. Condition now generally referred to as "hard pad disease."

Group II.—A demyelinating inflammatory encephalitis, not infectious to the ferret but transmissible to other dogs.

Group III.—An encephalitis, inflammatory, but *not* demyelinating. Apparently not infectious.

Group IV.—An encephalitis apparently caused by protozoal invasion of the brain.

Group V.—Miscellaneous conditions, the majority of nutritional or toxic origin. I would suggest that this group should *only* include those conditions in which histological brain lesions are demonstrable. This would eliminate most of those conditions misdiagnosed as encephalitis on the ground of their exhibiting nervous phenomena during life.

Let us examine these groups in more detail, more especially sub-group IB.

Group IA.—One important fact which the writers stress is that in all their experience, they have not isolated a strain of Laidlaw and Dunkin virus having true neurotropic characters. Even in dogs that have died showing nervous phenomena, re-injection of the virus even direct into nervous tissue of a susceptible dog will only give rise to ordinary distemper. The writers have also confirmed the features of the brain histology as described by Laidlaw and Dunkin, *i.e.*, nerve cell destruction with complete absence of demyelination and perivascular "cuffing."

Group IB.—From the observation that some ferrets which had been injected with material from canine encephalitis cases, when given a challenge dose of L. and D. virus, developed a *distemper-like disease in four of five days instead of the usual eight days*, the writers made the astute deduction that they had, by destroying their ferrets too early, been missing a virus differing from L. and D. virus. From this small beginning, aided by Dr. Margaret Sheitlin's observation that hardening of the foot pads in dogs affected with a mild distemper-like disease was a frequent indication of impending encephalitis, the writers have built up and described with the aid of veterinary practitioners a clinical and histological picture to which the use of the term "hard pad disease" has been applied. Personally, I am quite convinced that I have seen this "hard pad" condition before, but never in quite such a well-marked form. I must admit that never at any time had I the slightest suspicion that the hardened pads were of any pathological significance until my attention was drawn to this point. This exaggeration of the hardened pads which has occurred during these recent years may possibly have been brought about by the use of certain sulphonamides used to treat the tonsillitis and diarrhoea which so frequently occur during the early phases of this condition, and I should like to refer again to this point later.

To my mind, however, one of the most interesting and important features of this condition is the fact that the brain lesion is *essentially a demyelinating encephalitis*. For years now investigations into those distressing diseases of man associated with demyelination have been handicapped because no convenient experimental animal was available to which could be transmitted a virus having as an essential feature *and in certain circumstances* the power of setting up a demyelinating encephalitis. I feel that

the writers' plaint that they can only produce a thermal reaction by ordinary inoculation into other dogs, is a most excellent fact—since it will permit of an adequate search for and study of those various factors which may be involved in producing demyelination. For example, as I said earlier, there is already a good deal of clinical evidence to incriminate the early use of sulphathiazole and sulphamezathine as predisposing to the later development of brain lesions. When the outbreak was at its height, partly because my recovery results were bad, and partly on the score of expense, I started using A.P.C. mixture again for the primary fever-tonsillitis phase. At first I was suspicious that the lowered encephalitis development was pure chance, but as time went on suspicion developed into conviction that at least these two sulphonamides were to be avoided in these cases. On mentioning this to the writers, I was astonished to find that they also had the same suspicion and that their preliminary, but as yet incomplete experiments tended to confirm the same. There are quite a number of other factors that will have to be investigated in this connection—such factors as dietetic, vitamin and mineral deficiences, toxic factors such as overfeeding on agenised flour, predisposing factors such as excessive fatigue and nervous exhaustion, and precipitating factors such as intercurrent infections.

Group II.—I think I can support the writers' groupings better if I might refer, with your permission, to some of the conclusions and case records contained in a paper entitled "The Pathology of Disseminated Encephalo-myelitis of the Dog," by J. R. Perdrau and myself (*J. Path. and Bact.* (1930), **33**). In this paper we stated "It seems probable that the encephalomyelitis commonly referred to as 'the nervous form of distemper,' is not the result of the action of the specific virus of distemper on the central nervous system, but that the virus of distemper plays in this disease of the dog a similar role to that which an acute infection of *varying* aetiology plays in the causation of certain demyelinating diseases of man," and "Distemper is not apparently an essential antecedent of the encephalomyelitis."

The case records will be found as an appendix and I will refer to some of them later.

Group III.—Numerous cases of true canine encephalitis, showing varying degrees of the characteristic infiltration with small mononuclear cells of the meninges and perivascular spaces of the brain, with no demyelination, have been observed, and the case records of Dogs III, V and XVI are typical of this group. Although it is difficult, or at times impossible, to prove that these cases are associated with infections, I would prefer to leave out the writers' "apparently not infectious," and rely for the grouping on the histological picture alone.

Group IV.—An encephalitis caused by protozoal invasion of the brain. This condition would appear to be uncommon in this country. Dog X is an example of such a case. Clinically there would appear to be no way of diagnosing the condition.

May I refer back to a number of points concerning "hard pad disease," since this condition is the one that is of more immediate interest to clinicians at the moment.

In my opinion there would appear to be a large number of non-clinical cases in the field, comparable to the mild febrile reaction that the writers obtain after injecting the virus into a susceptible dog. My impression, and it is only an impression, is that these mild cases occur more frequently in older dogs and in dogs that have been immunised by vaccine-virus. Although I have seen only comparatively few recoveries after the occurrence of thickened pads and nose, there are again the same reasons for assuming immunity to distemper. As a suggestion, may there not exist a relationship between distemper virus and "hard pad virus" somewhat similar to that which exists between the virus of cow pox and human variola—some form of cross-immunity? In this connection, it is a curious fact that since "hard pad" has been so widespread, ordinary distemper has been comparatively uncommon.

"Hard pad disease" during the last two years has shown little tendency to obvious secondary invaders, and the clinical picture on that account has not been so distemper-like as on some previous occasions. Dogs I, II and VIII, whose brains showed the typical picture of "hard pad disease," will give some idea of how difficult differential diagnosis can be. It was such cases as these which converted me some 15 years ago to the idea that distemper, as occurring in the field, was made up of at least two entities, and that secondary organisms rendered the conditions at times clinically inseparable. Although it is impossible to prove now, the condition I described as "dry distemper" or "infectious brown mouth" (*Vet. J.* **92**. No. 11, 388 to 394), was probably a complicated outbreak of "hard pad" infection. In regard to treatment, I have tried most of the advocated lines and have not been very successful once nervous phenomena have been exhibited. The following constitutes my approach to an outbreak now:—

(1) Penicillin for the primary phase tonsillitis and fever. Absolute avoidance of sulphathiazole and sulphamezathine, even if tonsillitis is marked and diarrhoea severe.

(2) Full doses of aspirin in the form of A.P.C. mixture for at least one week. (A.P.C. = Aspirin dissolved in potassium citrate solution.)

(3) Full doses of Greenosan from commencement to recovery.

(4) *Absolute* and complete rest, however mild the symptoms, for three weeks or longer, if conjunctivitis is present.

(5) A generous diet containing meat offal with addition of several drops Crooke's halibut oil.

Finally, may I once more convey congratulations to Messrs. MacIntyre, Trevan and Montgomerie for their excellent paper on this controversial subject, containing information, which, in spite of reservations, I still consider will not only help us better to understand distemper in the field, but will also, I am convinced, lead to an advancement of knowledge in human pathology which will redound to the credit of both the veterinary and medical professions to which these writers belong.

APPENDIX

(*a*) Dog I of our series was a well-grown foxhound puppy, eight months old, which had never had a day's obvious illness until he came in contact with a case of distemper. A few days later he was taken suddenly ill with convulsions, complete loss of sphincteric control, galloping movements of legs and frothing at the mouth. These fits recurred every few hours until they were almost continuous, and the puppy was destroyed with chloroform on the fourth day of illness.

The principal *post-mortem* findings were as follows: a severe and fairly advanced bronchopneumonia, considerable swelling and engorgement of the mesenteric lymphatic glands and slight injection of the mucous membrane of the small intestine. The other internal organs were normal to the naked eye, as were the brain and spinal cord. Microscopic investigation of the central nervous system revealed an encephalitis and meningitis which did not differ essentially from those found in the other cases. In addition, patches of demyelination were found in the cerebellar peduncles and on the under surface of the pons, which, by their histological characters, were essentially of a chronic nature, and could not be differentiated from those found in Dog II (see Fig. 2), where the nervous symptoms had lasted two months. Had the demyelination been only four days old, one would have expected to find compound granular corpuscles (microglia) loaded with degenerated myelin. None was present, but one found instead a fibrous gliosis which had clearly taken considerably more than four days to develop. The nervous lesions in this case must, therefore, have antedated the distemper by an appreciable margin of time. The nature of the acute infection in this case was demonstrated by Major Dunkin by a successful transmission of the distemper to ferrets.

Fig. I

Dog I (killed four days after onset). Superficial patch of demyelination amongst arcuate fibres. Thick perivascular "cuff" and obvious meningeal infiltration.
Weigert-Pal and alum carmine. ×60.

Dog II. Border terrier bitch, aged two years.

Taken ill on December 10th with primary phase of distemper (many more dogs in this kennel developed the disease during the next few days). No untoward symptom presented itself till January 1st, 1928, when severe diarrhoea started. She gave birth to three live puppies on January 5th, 1928. These lived for 14 days and then died from distemper (19.1.28). On 19.1.28 the bitch had a convulsive seizure and when she recovered consciousness she appeared very frightened even in the presence of those she knew well. For the next two days she had similar convulsive turns, generally after food. On 24.1.28 she appeared much better and less frightened. On 28.1.28, however, her head became twisted round to one side, and when she walked she tended to go round in circles. On 30.1.28 nystagmus present; when placed on her legs she moved with gross ataxic movements. Whilst resting on her side there were rhythmic movements of both hind legs and one foreleg. There was still control of sphincters. She was chloroformed to death on this date.

Post-mortem Examination: Body wasted. Mild gastro-enteritis. Left-sided keratitis. No other lesion of note.

There were two other terriers actually living with this bitch. One developed acute encephalitic symptoms on 19.1.28 and was chloroformed on 20.1.28. The third terrier developed encephalitis 21.1.28 with myoclonic movements of left hind leg. This myoclonus increased in severity for about ten days and then slowly disappeared. During this time the dog was ataxic and amaurotic. Convulsive seizures became rather frequent, but after a month their severity lessened and the dog improved in health generally. The blindness which persisted is now (8.3.29) practically complete, but the hearing is good, the gait is still ataxic and a convulsive seizure occurs about every seven or eight days.

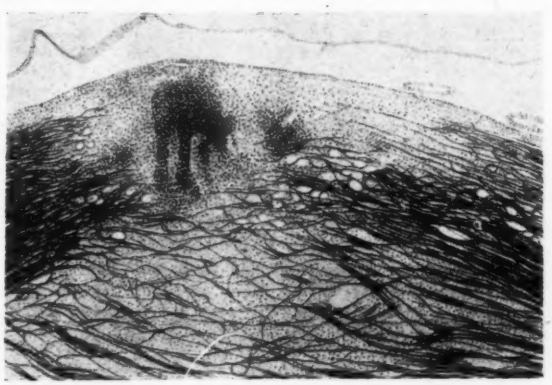


Fig. II

Dog II (killed two months after onset). Superficial patch of demyelination amongst arcuate fibres. Perivascular "cuffs," but no meningitis.
Weigert-Pal and alum carmine. ×60.

Dog III. Alsatian bitch, aged two years nine months.

In September, 1927, the bitch, although thin, was apparently healthy. Towards the end of October she commenced to lose her cheerfulness, became less obedient, showed lumbar pains and resented being handled. On 27.11.27 a convulsive seizure. From this date a slowly progressive ataxia developed, combined with amaurosis. During January, 1928, the limbs gradually became paralysed and the head was deviated to the right. Still feeding well. Muscles wasted considerably during February. On March 2nd muscles of throat appeared to become paralysed. Sphincters relaxed. Bitch destroyed by chloroform. No history of distemper.

Post-mortem Examination: Body very wasted. Stomach and intestines showed considerable areas of congestion. Spleen heavily pigmented. No other macroscopic lesions of note.

Histology: Inflammatory encephalitis. No demyelination.

Dog V. Male foxhound puppy, born June 16th, 1927.

No history of distemper. At end of October, 1927, said "to have looked odd and was very sleepy for a number of days."

27.11.27: Found to be holding his head on one side. Partial paralysis of hind limbs. Encephalitis diagnosed. Improvement took place during next few weeks. 29.12.27: Severe neuritic pains at back of head and over the lumbar region. Large doses of aspirin appeared to alleviate the acuteness of pain. During January the puppy improved generally so that on February 20th, 1928, he appeared normal except for slight stilted action of the hind legs. However, during a spell of cold weather at the end of this month, he again developed neuritic pains in his back and limbs so that he could hardly bear anyone touching him. March 10th: A more or less generalised neuritic condition present. Tried to bite through the skin of his hind legs. Mentally quite abnormal on this date. Would stand almost continuously in any peculiar position as though hypnotised. Slight inco-ordination of movements. Eyes apparently normal. Chloroformed to death 14.3.28.

Post-mortem Examination: No parasites found in ears or alimentary canal. Spleen enlarged and pale. Mesenteric and prescapular glands very large. Liver congested. No other naked-eye abnormality noted.

Histology: Perivascular "cuffing." No demyelination.

Dog XVI. Sealyham bitch, aged two years.

Strong, healthy bitch of placid disposition. Was mated on January 21st, 1929. A few days later it was noticed that she had lost her playful habits and, when at walk, all her usual interestedness.

About February 12th it was noticed that she showed very definite fear when out of doors. She would put her tail down and rush home screaming for no apparent reason. This condition became steadily worse, so that screaming fits occurred frequently during the day—the bitch running about the house and hiding under chairs and in corners. March 4th: Definite inco-ordination of movement noticed for the first time, with high-stepping gait. March 7th: Practically unable to stand. When placed on her feet would stagger forward and roll on to her side. March 9th: Destroyed by chloroform.

Post-mortem Examination: The only naked-eye lesion noted was a mild enteritis, especially of the duodenum with mucoid contents.

Histology: Perivascular "cuffing." No demyelination.

Dog VII. Sheep dog, four years old, male.

When this dog was first examined on April 12th, 1928, the owner complained that it was lifeless and disinclined to go for walks, an occurrence which was quite unknown in this animal. The history was that "the dog had been off his food for a day ten days previously, but had apparently recovered after the administration of castor oil."

On April 12th the temperature was 102·8° and considerable lethargy present; otherwise clinical examination did not reveal any abnormality. He was feeding well on this date also. On April 18th the dog became much worse. Temperature 104°. Respiration normal. Pulse slightly quickened. Great lethargy. Body wasting obvious. Slight paraplegia of hind legs. Intense conjunctival injection. On April 24th the dog became very restless, walking blindly round and round his kennel, always in one direction. There was complete amaurosis. When handled he evinced pain at back of neck and down all four limbs. On April 25th the dog was unable to recognise his owner's voice and was then chloroformed.

Post-mortem Examination: Some gastro-duodenitis present. No other naked-eye lesion.

Histology: Perivascular "cuffing." No demyelination.

Dog VIII. Cocker spaniel bitch, year-and-half old.

This bitch arrived with history of having had a mild attack of distemper six months previously.

She had been taken ill suddenly, the first thing noticed being the onset of convulsions. When examined temperature was 103·5°. Amaurosis with widely dilated pupils. Acute pain and tenderness at back of neck and down the left foreleg. No cough. No vomiting or diarrhoea. The "fits" recurred about every hour. After recovery from a convulsive turn, the bitch would wander about with inco-ordinate movements. Destroyed by chloroform at owner's request 24 hours after onset of convulsions.

Post-mortem Examination: Condition good, abundant subcutaneous and abdominal fat. No abnormality detected with the exception of mild gastro-duodenal catarrh. No parasites found.

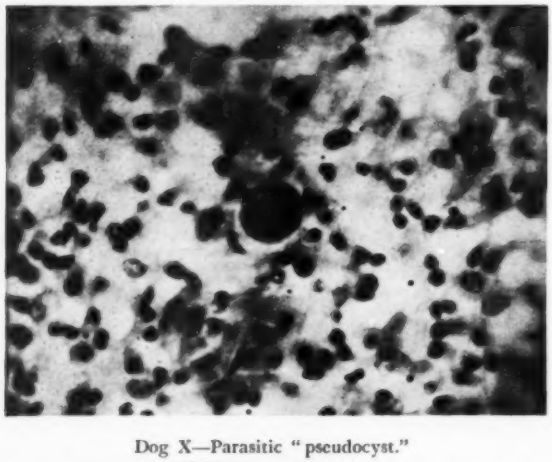
Histology: Demyelination and perivascular "cuffing."

Dog X. Parasitic encephalitis.

The microscopical findings in the case of one animal deserve special mention. It was that of a greyhound bitch (case X of our series), two years and ten months old, which developed an attack of distemper at a time when other dogs in the same kennels were affected by the disease. Five days after the onset of symptoms, ataxia was noted. During the next four weeks a mild degree of lameness was present at times, although the acute infection

itself was following its usual course towards apparent recovery. The fever and diarrhoea which had been present in the initial stages recurred, and the animal became rapidly worse. The head was deviated to the left and the ataxia increased in severity. This condition lasted two weeks approximately and then convulsions set in and the animal was destroyed with chloroform.

The naked-eye and microscopic findings in the central nervous system did not differ on the whole from those observed in the other case of this series. The usual severe and widely disseminated encephalomyclitis was present, but no demyelination was noted. At the posterior end of the right optic thalamus an area of necrosis measuring 5 mm. × 3 mm. was found. All the tissues normally presented at that spot were destroyed and no traces of them could be recognised. This area was invaded by a large number of small mononuclear cells and large compound granular corpuscles, together with an admixture of plasma cells and polymorphonuclear leucocytes. In addition, there were present collections of the "parasites" usually associated with the spontaneous encephalitis of the small rodents.



Dog X—Parasitic "pseudocyst."

* Dog XIII. Male mongrel Airedale, aged eleven months.

History of cough and malaise for two days in September, 1928. Noticed to be blind in left eye on January 10th, 1929. Within a week the dog was apparently deaf. He dragged his right fore and hind legs. When allowed his freedom he walked in wide circles. Feeding well and temperature normal. Gradual increase in loss of use of limbs. January 24th, 1929: Severe head tremors. Increasing amaurosis. No fever. Very slight conjunctivitis. Much thirst. Bowels normal. January 25th: Very irritable. Whimpering if disturbed in any way. January 26th: Not feeding. Destroyed by chloroform.

Post-mortem Examination: Acute gastro-duodenitis. Small areas of congestion in jejunum and ileum. Contents of small bowel mucoid. Large bowel apparently normal.

Dog XII. Histology: Demyelination and perivascular "cuffing."

(*b*) Our second observation relates to an outbreak of so-called hysteria in six puppies of a litter which came under my care. They all developed symptoms of an encephalomyelitis and two of them are included in our series (Nos. XIV and XV). They both showed characteristic lesions in the brain. A third puppy, owing to the owner's inability to look after it, was placed in an isolation kennel where it came in contact with a case of ordinary distemper, at a time when it still showed symptoms of the nervous affection. This puppy contracted distemper a week later and passed through a typical course with the characteristic primary and secondary rises of temperature. There was, however, no exacerbation of the nervous symptoms, and the puppy returned to its owner after being apparently well for four weeks. This animal is now said to be nervy and peculiar, but otherwise apparently fit. Had the encephalitis in this case resulted from an unsuspected attack of distemper, one would have expected this animal to have been immune to reinfection.

Dog XIV. Male foxhound puppy, aged eight months.

History of peculiar nervous phenomena over a period of two months affecting all the puppies (six in number) of one litter. These young dogs were in almost daily contact with other young dogs, none of which has so far developed any nervous symptoms. The first abnormality reported was the constant eating of turf and grass which they would pull up in large amounts. Treatment for ascarides was carried out at the time with oil of chenopodium, but, as repeated treatment did not prevent the pica, it was assumed that some other factor was the cause of the gastric irritation.

This hound had shown progressive blindness, attacks of hysterical howling and convulsive seizures. After each convulsion would stagger about for a short time and then become fairly cheerful again. Feeding well and in good bodily condition. Bowels normal. Died suddenly in a convulsion 27.1.29. No parasites in the ears.

Post-mortem Examination: Acute gastro-duodenitis. Large number of ascarides in small intestine, the contents of which were mucoid and heavily bile-stained. The large bowel was normal in appearance and in contents.

Histology: Perivascular "cuffing." No demyelination.

Dog XV. Foxhound puppy, litter sister to No. XIV.

History of repeated hysterical turns. Would suddenly assume a vacant stare and commence nodding rapidly. If forced to move, she would do so in an ataxic fashion. Recovery after several minutes. The sight of food or any form of excitement was enough to start an attack. A sudden convulsion followed by death. No parasites in the ears or alimentary canal.

Post-mortem Examination: Acute gastritis. Patchy areas of inflammation in small intestine. Small bowel contents mucoid. Large bowel normal.

Histology: Perivascular "cuffing." No demyelination.

* * * * *

General Discussion

The first speaker in the general discussion was Mr. W. Mansey, of the Serum Institute, Cairo, Egypt, who said that it afforded him great pleasure to have that opportunity of congratulating Mr. MacIntyre, Dr. Trevan and Dr. Montgomerie upon their excellent paper on a very interesting subject.

Encephalitis in the dog, as it had been shown, covered several syndromes. What interested him was that form of encephalitis caused by the distemper virus.

Encephalitis in distemper cases might be explained in two ways:—

(1) The well-accepted theory that it was a pathological affection of the central nervous system as a complication of the general morbid features of the disease, depending on the individual susceptibility.

(2) Although research workers had not been able to isolate a neurotropic strain from nervous cases of clinical distemper, yet there was some possibility that highly neurotropic immunologically distinct strains of distemper existed.

Following experimental inoculation of the virus, nervous symptoms were very rare among dogs or ferrets. There appeared to be some factor which was missing, for, when the viscero-tropic virus was inoculated into dogs or ferrets, it did not produce the "nervous" distemper.

Vaccination against distemper with the present vaccines prepared from spleen tissues or by the virus-serum method, did not prevent the occurrence of encephalitis in dogs, although the percentage of infection was lowered against alimentary, pulmonary, cutaneous or ocular forms of the disease. The explanation of this seemed to be, that these vaccines were prepared from a viscerotropic virus which had been passaged through susceptible ferrets or dogs, thus concentrating the viscerotropic affinity of the virus, with the result that vaccinated dogs would resist a further infection of the same viscerotropic virus, as had been proved in potency tests applied by the firms producing these vaccines.

Anti-distemper serum was also prepared from dogs immunised against the viscerotropic strain. This explained why, in exposure to natural infection, certain vaccinated dogs developed nervous symptoms.

Vaccination of dogs in Great Britain by the viscerotropic strain vaccines had to a certain extent aborted the appearance of the usual alimentary, pulmonary, cutaneous or ocular types of distemper, but at the same time the natural prevalent virus attained a neurotropic affinity which was enhanced by the diminution of the viscerotropic effects. This explained the increase in nervous cases of distemper and it would increase, unless there could be produced a neurotropic vaccine to control it.

Mice could be infected with canine distemper virus following intracerebral injection of a known passaged strain, such as that used by him in Cairo in 1944, or that he received from the Wellcome Laboratories.

Symptoms: After the usual incubation period, which was nearly fixed to a week interval (six, seven or eight days) the following

* Dog XIII is probably a case that would be classed under Group II.

symptoms rapidly appeared, ending in death within 24 hours in 80 per cent, of the inoculated mice: rough coat, dullness, irritation of the eyelids, watery discharge from the eyes, which later became swollen and inflamed. Usually within a few hours the eyelids were partially closed with a sticky discharge which was sometimes purulent; later, in the third passage, nervous fits began to appear. In these fits severe tetany of the extensor muscles of the hind legs almost always developed, leaving the legs and feet fully extended in a backward direction, with the solar aspect of the feet upwards and the digits showing a characteristic semi-contracted appearance On the ninth passage opacity of both eyes was noticed. After six to 12 hours from the appearance of the symptoms, regularly occurring nervous fits gradually appeared. Any stimulus such as touching or handling the mouse in an upside down position, almost invariably increased the tendency towards the development of a nervous fit. These fits occurred regularly and might last for a few minutes, after which the mouse recovered, only to develop another fit after an interval of 15 to 30 minutes. Numerous fits might occur before the mouse died, and death usually took place during one of them.

The use of the mouse as an experimental animal for work with distemper virus should be an advantage in that it would no longer be necessary to adopt the strict isolation procedures required when dogs or ferrets were employed.

The short incubation period—about five days—with the Cairo strain and its difference from that of the Wellcome strain—about seven days, but never less than six days even after the tenth passage—might indicate a difference between the two strains. The rapid development and the violence of the nervous fits with the Wellcome strain after the third passage in mice was another difference from the Cairo strain with which the nervous fits were mild and did not appear before the 13th passage.

The Wellcome strain had a marked tendency to neurotropism. Although in mice the virulence was increased by passage, yet this passaged virus had no harmful effect on inoculated dogs or ferrets by the usual routes of inoculation. Intracerebral inoculation into four young ferrets developed in one of them a transient nervous fit, a maximum thermal reaction of 105·4° F. being recorded. The animal soon recovered.

It was highly probable that there was more than one strain of virus in this country, each manifesting itself by different clinical symptoms in various outbreaks.

It was therefore preferable to collect infectious materials from different outbreaks, and inoculate them intracerebrally into mice; thus by pooling the mice brains, a polyvalent neurotropic attenuated virus vaccine would be obtained.

Mr. Ian McIntyre (Edinburgh) congratulated the authors on their most interesting paper and said that he wished to acknowledge that it was Dr. Montgomerie who first drew their attention to "hard pad disease" in the spring of this year.

The observations he was going to give were, therefore, collected over a short period of time and were based on only 30 definite clinical cases of the disease and *post-mortem* examination of ten of these. They had been accumulated with the help of his colleagues.

The chief clinical differences from the picture described by the authors were the complete absence of fits in all but two of the 30 cases despite the fact that over 20 were known to have died, and a much more severe type of pneumonia. The clinical examination of the chest was misleading. The respiratory rate was increased to 60 to 80 per minute. Ausculation revealed only an increase in the bronchial and vesicular sounds which could be heard evenly over the whole area of ausculation. In advanced cases there was a marked decrease in resonance on percussion over the upper third of the area.

On *post-mortem* examination they had so far been unable to find evidence of demyelination in the brain, but then there had been no clinical cases of "fits" to examine histologically.

The picture presented by the lungs varied from small bluish patches scattered here and there in the milder cases to large areas of both lungs being completely consolidated. Histologically, the picture was that of an interstitial pneumonia with massive occlusion of alveoli as a result of proliferative or infiltrative changes in the alveolar walls.

Although the above observations were of limited value he hoped that they would help those studying it to form a general picture of this disease in its different aspects.

[On November 4th, Mr. McIntyre wrote: "Clinical Impressions—Over the last two months it would appear that the cases under our observation have been of a milder type with few deaths and a continuing small percentage of nervous symptoms."]

Mr. H. B. Parry (Bury St. Edmunds), after thanking the authors for their paper. and Mr. Pugh for his most informative opening remarks, said that as a comparative newcomer to this field he felt some trepidation in making any comments. There were, however, one or two points which interested all of them and which he thought should be clarified now in order to avoid confusion.

He concurred strongly with the view that nutritional disorders should not be termed encephalitis, which should be confined to inflammatory disease. There were probably many other nervous diseases in the dog which were neither of nutritional origin nor virus infections, and which required definition.

With regard to the role of so-called nutritional deficiencies in the development of nervous lesions, he had to take exception to the view that one encountered convulsions and neurasthenia with deficiency of vitamin B_1. In experimental cases of acute deficiency of vitamin B_1 in the dog there were no nervous signs primarily due to defects in the central nervous system. The authors of the paper had suggested that one of the reasons why nervous diseases should be so prevalent at the present time was because of the general lack of nutrition in the dog population, but as far as the speaker was aware no accurate work had been carried out in this connection on dogs. However, in the last five years there had been very careful tests with mice, using poliomyelitis virus, and using encephalomyelitis virus with chicks, and the results had been most interesting. Virus infection was much *less* likely to cause damage to the central nervous system in the deficient animal than in the animal which was adequately nourished. Chicks on a vitamin B_1 deficient diet infected with virus would show no signs of leg paralysis, whereas if they were on an adequate vitamin B_1 diet they would develop leg paralysis. With virus infections it was likely that the ill nourished would be less liable to show residual nervous defects than well nourished animals. It would be interesting to have more clinical evidence on the use of large doses of vitamin B_1 therapeutically.

Concerning treatment he wished to endorse Mr. Pugh's remarks about the value of absolute and complete rest. The remarks given by that contributor had been most illuminating. The outbreak of poliomyelitis in the previous year had shown conclusively that many of those who developed residual paralysis often had carried out severe physical work during the initial febrile illness.

Mr. W. L. Weipers (Glasgow): I should like to say at the outset that without the co-operation of Dr. Montgomerie and his colleagues, I could add little to a discussion on the differential diagnosis of the different types of canine virus infections. I have always maintained that most research teams into naturally occurring disease should contain clinical workers. I have suggested that Dr. Montgomerie should indicate what type of material he wishes from clinicians, how most such material is useless without adequate case history and how much its value is enhanced if it is part of a series likely to be submitted of an epidemic, particularly an isolated epidemic such as occurs, for instance, on an island or in the centre of sheep country where the disease has not appeared for years and affects adults and young dogs. Selected material from one such outbreak, I maintain, is more enlightening than hundreds of cases of street distemper and we know that Dr. Montgomerie and his colleagues are inundated with such material, much of it useless.

In a selected number of cases which included one typical epidemic, I kept a fairly exact clinical record and in most cases where the animal died I sent selected material to the Wellcome Laboratories for ferret and dog inoculations and in most cases, material for microscopical examination. In many of these cases, we had a clinical history and in a few we were able to trace the epidemic relationships and correlate the result of animal inoculation. My interpretation of the facts probably differs slightly from that of the Wellcome team because of my clinical viewpoint.

I wish to congratulate the authors on bringing forward this paper at this interim stage. It would have been much easier for them to have waited until their work was further advanced, but the subject is of such vital importance that the profession has pressed for this preview and I, personally, believe that this course should allow practitioners to aid with selected material. Major Pugh's contributions on this subject had a similar basis and his earlier recorded work forms a substantial part which will have to be related to present research, particularly on the micro-pathological side.

Our conception of virus and virus diseases is inevitably based on our knowledge of bacteria and bacterial disease; but, as evidence accumulates, I think this will require much drastic revision. The viruses show many differences from bacteria and while many virus diseases show the same stable character and reappear in the same form year in year out—and, as far as can be ascertained, do not change over a period of decades—others are much more unstable, and apparently new virus diseases spring up—or new variants—and these sometimes sweep over the globe in virulent form and the causal organisms either die or become so modified as to be unrecognisable. There has been some definite evidence that serial inoculation with single viruses can produce uniform results over several generations, to be followed by what cannot otherwise be described than as a complete variation manifested by totally

different symptoms. These new characteristics may or may not be stable.

In other well-established virus diseases, the age incidence might shift over a period of two decades from one age group to another, or the disease might so change in its clinical character as to become unrecognisable; or the parasite might find a new host. The reason for this might be found in the ecological field or might be in the stabilisation of the parasite-host relationship. When one considers the possible methods of reproduction of viruses it must be realised that even in the light of present limited knowledge, such methods cannot possibly be on the same physical basis as any known cellular division because of its admittedly different structure, and it is reasonable to suppose that it is more likely to follow processes similar to nuclear divisions.

It is known that viruses (as evidenced by the effects they produce) can be changed and whether this is due to a change in the nature of the virus (which might or might not be stable) or to the selection of something similar to genotypes, is so far a matter of speculation. I think the weight of evidence suggests that there are probably certain predictable variations which in certain surroundings will survive almost to the exclusion of the original type. In certain circumstances the re-establishment of the original type could take place when external circumstances favour its development. Perhaps some day someone will work out a law of Mendel that will be applicable to viruses generally. A simpler explanation might be that put forward by Burnett and Beveridge—that variants occur in a proportion apparently equal to the mutation rate in any species and that the apparent difference in speed of the occurrence of variants can be explained by the enormously accelerated reproduction rate of these parasites.

If "hard pad" is a new variant of distemper Laidlaw Dunkin virus, then one would expect some antigenic relationship. I think there is considerable evidence to show that this does exist. It also follows that if the variant has an enhanced survival value, it could also be expected that it would in time tend to supplant the earlier type, and in my opinion there is some evidence for this. If my suggestion is right—that the virus in its present form is unstable—its further development is unpredictable; but one would have to consider two possibilities: one, that it would cease to maintain its present survival value and the earlier type would reappear; or two, that it would possibly find establishment in a new host and man cannot be excluded from this possible relationship.

About two years ago, typical "hard pad" cases appeared in my district and at that time every case was fatal. Most dogs developed fits and chorea. Some of the dogs had been inoculated with virus vaccine against distemper. At that time, we considered that we were dealing with a recently introduced disease, but in the light of subsequent happenings, I have come to the conclusion that the causal organism is a variant of ordinary distemper virus (Laidlaw Dunkin) and that one of its characteristics is a capacity for further variation—it appears to lack stability.

From discussion with colleagues in other parts of the country, I gather symptoms vary enormously, *e.g.*, Mr. McIntyre of the Royal (Dick) College has stated that he had never seen nervous complications following "hard pad disease" and he is in practice less than 50 miles from my home. Since the disease first became apparent, there has been a diminution in virulence. By the late winter of 1947, quite a number of cases were recovering; mortality was about 50 per cent. By the spring of this year, many cases that did not recover died without showing any nervous symptoms. It may be that they did not live sufficiently long for their development, but none of the survivors developed these symptoms. The course was fairly typical—a seven day broncho-pneumonic period (which appeared to respond to penicillin) was followed in two or three days by a relapse; respiration rate was accelerated, but percussion failed to reveal a dull note. Auscultation was negative—only friction-sound, due to the greatly increased respiratory rate, could be heard; the heart was fast at first and the pulse later showed a running-down character and, in certain cases, became as slow as 60 prior to death. There was a peculiar vibrant characteristic when the hand was held over the area of the heart. Penicillin showed no obvious results at this stage but alcohol frequently administered appeared to be beneficial. A number of cases made an uninterrupted recovery from this stage and showed no permanent heart disease. *Post-mortem* examination of cases that had died showed a solid mottled grey unusual condition of the lungs (these sank in water); cases that were destroyed 24 hours before death was likely to have occurred, had usually solid areas along the borders of the lungs. I suspected that the consolidation was probably circulatory in origin and that death resulted from heart failure. I did not have an opportunity of having such lungs sectioned but concluded on clinical grounds that the condition was in the nature of an oedema. I presumed that the entire lung would have given a dull note on percussion immediately prior to death, but I was unfortunate in never once being present at this stage. I have since discussed the matter with Mr. McIntyre and his Edinburgh colleagues and they recognise a similar syndrome. They have had lungs sectioned in the Pathology Department and I understand that the condition was not an oedema but a pneumonia. During the late summer, the type of case has been more severe but this may be one of the normal fluctuations that are encountered during any epidemic. As the result of studying the co-ordinated evidence, clinical and micro-pathological and experimental injection, I have come for the time being to the following conclusions:—

(1) That "hard pad disease" and distemper (Laidlaw Dunkin) virus disease are closely related.

(2) That a dog immune as a result of an initial attack of "distemper" as it occurs in this country in the field or street will not succumb to a natural attack of "hard pad" virus.

(3) That distemper serum probably gives some limited protection against naturally occurring "hard pad disease" if given sufficiently early or during the incubation period.

I think some of the apparent failures in the field arise from the fact that the onset of "hard pad disease" is so insidious and there fact that the onset of "hard pad disease" is so insidious and that there is often no temperature, making is very difficult to know if indeed serum has been given sufficiently early.

I also consider that, contrary to what one would expect with a homologous serum, distemper serum only protects against the natural disease for 12 days and thereafter there is a rapid loss of immunity. I base my observations certainly on a very few cases but, particularly where a known healthy dog was introduced into an infected house and was immediately given serum, the dose being repeated at ten-day intervals, the usual course was complete protection or the development of only mild disease with recovery.

(4) That in my district I appear to get few if any cases of typical Laidlaw Dunkin disease.

(5) Dogs that have been injected with vaccine-virus B.W. have a better chance of recovery from "hard pad disease" than uninoculated dogs, but a distinct proportion will succumb.

A point I intended raising was the relationship of fox encephalitis, but I am glad to hear from Dr. Montgomerie that this is still another clinical entity and does not fall into any of his groups.

There is a vast difference between laboratory distemper and field or street distemper. At no time in the last 20 years would I have classed street distemper as a mild disease with a percentage of fits comparable to Dr. Montgomerie's figures. My percentage has always been much higher—100 per cent. in some outbreaks, rarely less than 10 per cent.

I think group III can be recognised clinically and is not likely to be confused with I or II. There is some evidence that this disease may be transmitted to ferrets, causing death, without previous symptoms in three or four weeks.

I think group II is probably of fairly rare occurrence, although from continental literature it appears to be common there. I have only once sent material to Dr. Montgomerie which proved microscopically to correspond to this type and clinically it was not characteristic.

It is interesting that the injection of "hard pad" virus will not produce the disease in susceptible laboratory dogs as it does in ferrets, but I cannot think that an essential prerequisite for its established in the field is low nutritional level, etc., as I have seen the disease appear in every conceivable circumstance with good, bad and indifferent management. It merely stresses the difference between street and laboratory disease and I would suggest the extension of the experimental methods to the field.

It is a matter of great importance that in an outbreak of "hard pad disease" in a kennel, some susceptible dogs develop the disease without at any time developing typically hard pads that can be recognised macroscopically. In the ordinary surgery examination of such cases (without the epidemiological background) this fact would render any attempt at clinical classification extremely inadequate.

It seems difficult to reconcile an antigenic relationship of distemper and "hard pad" diseases with the diversity of microscopic lesions in the central nervous system of the two diseases but it could be explained if it could be proved that Laidlaw Dunkin virus has not been responsible for naturally occurring disease in this country for the past few years.

As evidence accumulates on the micro-pathology, the frequency and distribution of the various types will be readily established and probably many of the apparent contradictions will be resolved.

I again wish to thank the authors for what I believe to be a large step forward in this field and hope to see a further contribution from them soon, even if it is only a further interim report. For my own part, I realise that I have made some statements that cannot be substantiated and that I am sure will have to be ultimately revised; but I do so in the knowledge that they are useful parts of a working hypothesis and I will willingly discard any part of it that new knowledge renders untenable.

Contributions Subsequently Received

(In Response to "Open" Invitation Extended Through *The Veterinary Record*)

Mr. A. L. Duckett (Cardiff): It has been reasonably well established, by various workers, that this disease is caused by a virus distinct from the true distemper virus of Laidlaw and Dunkin.

Reviewing a large number of cases of this disease, a classification into three main groups appeared to be possible, as follows:—

(1) Gastro-intestinal upset leading to a persistent diarrhoea with, usually, a fluctuating high temperature. Conjunctivitis is a constant symptom, and fairly early in the course of the disease, the pads harden. In the later stages, convulsions and chorea develop and death results after a prolonged illness. The appetite is usually fairly well maintained.

(2) Tonsillitis is the first sign of infection. The animal may recover quickly from these symptoms and then relapse, but more commonly a slight temperature persists for several days, without further sign of illness. The pads gradually harden and this is followed by the usual convulsions and chorea. Quite commonly chorea develops before any convulsions are noted.

(3) Slight hysteria or minor convulsions in an otherwise normal dog. Vague respiratory symptoms may follow, with a watery nasal discharge, sometimes becoming purulent. Hardening of the pads follows, to be succeeded by the usual nervous sequelae.

The groups are not always distinct, *e.g.*, pneumonia frequently occurs in Group I. Indeed, a degree of pneumonia appears to be an almost constant factor in most fatal cases.

I have formed the conclusion that the "hard pad disease" is highly infectious, but that in itself, it is not able to produce clinically recognisable disease and remains dormant in the infected animal. When a dog infected with this dormant virus has its vitality lowered, the virus becomes active. The early symptoms depend entirely upon this secondary exciting factor: it may be a disease, *e.g.*, tonsillitis, puppy fits due to teeth or worms, or inoculation of an apparently healthy puppy with the Laidlaw Dunkin virus during immunisation against distemper.

The duration of the disease varies enormously, but tends to be of shorter duration in young puppies. Adult dogs are not immune but have a much better chance of recovery.

Treatment.—To be effective this must be started early in the disease. Unfortunately, early diagnosis is very difficult. The only really constant feature is the hardness of the pads, and even this does not invariably develop. Once the pads have hardened it is usually quite useless to attempt treatment.

It has been my practice to give 2 c.c. doses intramuscularly of "Plexan" liver extract on two consecutive days to suspected cases, and to follow this with the same dose at two or three day intervals until a definite improvement takes place, when the dose is given once weekly until recovery is complete. Feeding raw liver by mouth also appears to be helpful. Symptoms are controlled as they arise, avoiding the use of the sulphonamides which appear to be harmful. Penicillin is of doubtful efficacy even when pneumonia is present as a prominent symptom.

If administered early, the "liver" treatment will not prevent the pads developing a fairly marked degree of hardness but it does give from 40 to 50 per cent. recoveries in all but the very young.

All my cases were seen in practice in Portsmouth where the disease is very prevalent. Since moving to Cardiff I have not seen a case of this disease in two months of practice, nor have I met a practitioner (qualified!) in this area who is familiar with the disease.

Mr. H. Raynor Hewetson (Southport): I was unfortunately prevented from attending the discussion on this paper and consequently do not know what points were raised.

Time and space will not permit reference to more than a few points in the paper, and I hope I avoid mostly what has already been said. One which I hope I am repeating is the appreciation and gratitude of those extensively engaged in small-animal practice at the work done by the authors, the presentation of this, what we may call an interim report, and the promise of the pursuit of the investigations until their objectives are attained.

In 1934 I first became disturbed by a sequence of cases apparently infective, exhibiting all or any of the nervous symptoms described; fits, hysteria, chorea, paraplegia, paralysis, dementia, coma, ataxia, amaurosis, without any prodromal symptoms of distemper or any other illness. By 1937 I had been subjected to an epidemic of such cases, a number of which arose in my own kennels, and the mortality was about 99 per cent. In one instance, a dog from a kennel of mastiffs came to my hospital for an entropion operation and stayed one night. About ten days later this dog commenced to drag its hind legs, rapidly became paralysed, developed fits and finally dementia, and was found one morning in a coma with one fore leg completely chewed off. One by one the other dogs in these kennels contracted the disease, developing a variety of nervous manifestations until in ten weeks 21 out of 28 dogs from four months to seven years were dead. The seven dogs which remained were unaffected throughout, and were the only seven which had previously been immunised with Laidlaw and Dunkin vaccine-virus. *Post-mortem* examinations of a number were carried out at the University of Liverpool without any definite result, and tissue and swabs forwarded to Beckenham for ferret tests were negative for distemper virus. All the affected dogs ran high temperatures; they mostly ate well, even ravenously; a lot had diarrhoea and all had marked congestion of the vessels of the sclera whether accompanied by conjunctivitis or not. The combination of fever, ravenous appetite and congestion in the sclera is in my experience a certain indication of the development of encephalitis. A similar distressing history covered all the dogs in my own kennels at the same period with cases here, there and everywhere amongst those patients and boarders which had gone home before symptoms commenced. I envisaged my reputation and practice disappearing and myself in dungarees with a brush, shovel and cart tidying up the streets.

For some time after this I made a practice of sending the carcases of selected cases to Dr. J. R. M. Innes, then at the Institute of Animal Pathology, Cambridge; and material taken during life to Dr. Montgomerie at the Wellcome Research Laboratories for tests of distemper virus, and tried to correlate the symptoms during life with these reports. Though this ultimately reached a dead end without much success, certain facts of interest emerged:—

(1) Though all the cases which had symptomatic encephalitis during life, apparently contracted by infection from other dogs, showed a meningo-encephalitis on *post-mortem* examination, the overwhelming majority exhibited no typical distemper symptoms during life and material from them failed to infect ferrets.

(2) The brain lesions could be roughly grouped histologically but not correlated to symptoms during life. The largest histological group corresponded largely with Group Ia in the paper but none of the animals was noted to have "hard pads."

(3) It was obvious that more than one neurological entity occurred in the dog which could not be lumped under the label "distemper" or "post-distemper encephalitis."

(4) Though another causal virus or viruses seemed certain, there appeared to be some relationship between them, since dogs immunised against distemper virus usually escaped in an encephalitis epidemic. In fact it is as recently as three years ago that I had my first case of encephalitis in a dog immunised against distemper and only three others since. These all died. In 1939, Dr. Innes, having had no opportunity of doing transmission work, and being unable to pursue his investigations further, forwarded all his specimens, sections, slides and data to Dr. H. Scherer of Antwerp, in the belief that he would be able to pursue the investigations under suitable conditions. All the material disappeared in the German Occupation of 1940, but some of Dr. Innes' findings are reviewed in a paper he presented to the Lancashire Division in 1940.

I have devoted some space to this reference to show that canine encephalitis has been a headache to practitioners for a large number of years, even at a time when ample supplies of proper food were easily and cheaply available. Although I would be the first to agree that good diet—which should be largely meat—and good management, are of prime importance in the development of sound health and resistance to disease, I cannot accept the statement that impaired nutrition is an important factor in the causation of canine encephalitis other than those in Group V. I have read carefully the symptoms described under the different groups but am quite unable to classify my own cases to any such degree. The majority of cases most closely resemble Group II except for the feature described as characteristic, the tendency to walk in circles. A few may, but not the majority, yet blindness, paraplegia and chorea are common and this would seem to exclude them from Group Ia, though they are patently infectious. I think I take reasonable care in my examinations and for some time have made a careful examination of the pads of all animals under remote suspicion, yet not until this last week have I encountered the hard pad in any cases, and I have now seen only two. The most baffling feature to me is that when I encounter an outbreak in a kennel with, say, 10 to 20 cases, practically every case will present a different set of symptoms, physical and nervous. Similarly cases which appear to me marked for certain death or destruction sometimes make impossible but complete recoveries, and others whose symptoms are slight will quickly pass into a coma and die. It is this mixing of symptoms and results in one outbreak in one kennel which baffles my attempts to make, or readiness to accept, a satisfactory classification of symptoms related to a specific disease. I have had a number of assistants who have come to me from the South, and/or who have left me to go South, and they have all told me the encephalitis they have seen with me appears to be different from and worse than what they encounter in the South of England.

This angle may be worth pursuing though conditions for many years have prevented me devoting any time to investigating cases beyond their natural conclusion.

Treatment is of primary importance to the practitioner, and I think the authors might have been bold enough to mention by name the sulphonamides which they consider a factor leading to the development of hard pads. Personally I think the sulphonamides have a very limited field of usefulness in canine practice, and none at all in the treatment of distemper and allied conditions, and I feel almost the same about penicillin. When I compare the small degree of success which follows their appropriate and proper use with the waste and damage done by their inept and often incorrect administration, I think canine practice would, on the whole, be better if they were completely discarded. Their success in wounds and operative cases is leading to bad and careless surgery, where the operator feels he can get away with anything. The objective of the authors is the one we all look for, the supply of effective prophylactics and anti-sera. I have entirely abandoned the use of anti-distemper serum as a therapeutic agent even in the earliest stages, having had no success with massive doses at prohibitive cost commencing on the first day of malaise in cases whose course later was typical of true distemper. In any therapeutic remedy this question of cost will always have to borne in mind, because not many owners can afford the money for massive and repeated doses of expensive sera, and vitamins such as aneurin, and liver extracts. Do the authors suggest the feeding of flesh and offal to dogs running high temperatures and suffering from diarrhoea? They exclude crude cane sugar molasses in these circumstances, but in any case, how is this obtainable in these days? Do the authors suggest the forcible feeding of sick animals whose natural tendency is to resist food? I fully agree with them that any measure of treatment should be directed towards assisting the natural recuperative processes, and I would add that if you are not sure that what you are going to do is going to assist, then for Heaven's sake do nothing to hinder.

In the absence of proved useful remedies I pin my faith in hygiene and nursing and not too much of either. If a sick dog is disturbed every four hours to be cleaned up, be given what medicines are thought desirable, and offered food if it desires it, it is being worried enough, and for the rest of the time I insist on non-interference. I never have a sleeping patient wakened for anything. Normally I use nuclein and vitamins as routine, kaolin and bismuth salicylate for diarrhoea, heroin for distressing cough, aspirin or codein Co. for restlessness and fits, supplemented by bromide and chloral if necessary, normal saline for dehydration. Barbiturates are useful only to soothe the pathway to death. Some cases may call for injections of vitamin B, liver extract, atropine, arsenical preparations and so forth, but these should be reserved for the suitable time in suitable cases, and not just pumped into all and sundry on imaginary pretext. For food I like above all things milk and eggs, and prefer white fish, fowl and rabbit to heavier meats, and am not frightened of a certain amount of bread or biscuit if desired. Not all dogs are allergic to whatever toxin occurs in agenised flour and many patients have already been eating it for a long time previous to illness without ill effect. I do not see a close relationship between the toxic hysteria of Mellanby and an infective encephalitis, and in any case his findings are not fully borne out by the histories and treatment of naturally occurring hysteria in every-day practice.

The Reply

Reply to the discussion was made by Dr. Montgomerie, representing also his co-authors, as follows: I know you will readily agree that Major Pugh's opening was an excellent introduction of the discussion, particularly since he was able to base his remarks on both a wide experience and much investigation. It is satisfactory to find that he could give approval to the classification which we have advanced and, in fact, confirm some of our contentions.

He has expressed his view that at least some of the so-called breakdowns in immunising against distemper were really the result of a failure to appreciate the existence of these other diseases of the dog. In this connection we would wish to say that an explanation can be offered for these occasional cases in which what some practitioners call a distemper-like disease appears within a few days of the injection of distemper virus in immunising. In some of our experiments we have observed that dogs which recovered following the injection of "hard pad" virus have shown a severe reaction within 24 hours of an injection of distemper virus. Blood taken from these dogs at the time of this reaction has infected ferrets in the manner characteristic of "hard pad" virus—an incubation period of over 15 days, a relatively mild, long progressive illness and the appearance of hard pads. We cannot, then, but feel that some of the field reports referred to above have arisen from the injection of distemper virus in a dog already "carrying" "hard pad" virus.

Major Pugh has rather challenged our action in placing the variety of conditions in the group which we have called No. V. We accept his criticism as logical and just, but in some of these nutritional and toxic conditions there is a brain lesion of sorts which, so far, we have not attempted to describe. We felt we had to mention these conditions and, tentatively, put them in a miscellaneous group. They did account for quite a proportion of the cases clinically diagnosed as encephalitis.

Major Pugh has expressed his belief that he has seen "hard pad disease" some years before we first recognised it as an entity. Our perusal of his published articles and illustrations leads us to agree. We did not intend to leave the impression that "hard pad disease" did not occur in Britain before we drew attention to it and we would point out that our acceptance of the standard practice of observing the injected ferrets for only 15 days was probably the main reason why we did not detect "hard pad" virus prior to 1946. Even so, we would contend that the disease was of relatively rare occurrence prior to about 1944. Perhaps there is something in the age-old contention that one only finds a thing if one is looking for it.

We were most interested when we found that Major Pugh's clinical experiences confirmed our view that the early use of at least some of the sulphonamide drugs predisposed to the full expression of the effects of "hard pad" virus infection. While the groups of dogs which we had on these drugs and then exposed to virus were not large, our observations suggest that the use of sulphathiazole and sulphamezathine was more dangerous than that of sulphapyridine and sulphaguanidine. All the dogs on the two former drugs developed convulsions or other signs of encephalitis ten or twelve days after the injection of the virus.

Although Major Pugh had published his conclusion that distemper is not an essential antecedent of encephalomyelitis in the dog, few practitioners appear to have accepted this. We hope that with the evidence now before the profession there will be less tendency to accept these nervous conditions in the dog as a post-distemper syndrome.

Major Pugh seemed a little surprised that we should characterise our Group III cases as apparently not infectious. Our work on this group has been somewhat limited but, so far, we have failed to transmit this type.

We would readily agree with the contention that quite a number of non-clinical cases of "hard pad disease" do occur in the dog population, but we are not prepared to accept the suggestion that the virus of this disease and that of dog distemper have a relationship similar to that which exists between cow pox and smallpox. In the latter case there is a complete cross-immunity, whereas the furthest we can go is to say that immunity to "hard pad" virus does not protect against distemper virus although there may be some protection against "hard pad" virus in the distemper immune dog.

All must have listened to Mr. Mansey's contribution with considerable interest and some may have accepted his explanation of the occasional occurrence of distemper encephalitis in the immunised dog. We had no knowledge of his findings until we attended the Congress and, had we not to reply to the discussion, we would have preferred to reserve some of our points for personal discussion with him.

We believe that Mr. Mansey is not familiar with the true position relating to the field use of distemper prophylactics in Great Britain. Apparently he assumes that in the vast majority of the so-called failures to immunise, encephalitic symptoms appear without the animal having shown the typical distemper syndrome. This is not in accordance with our experience, for where distemper does occur in an "immunised" dog the course of the disease is typical.

We have no experience of two types or phases of the distemper virus—the one viscerotropic and the other neurotropic. The strains isolated from brain tissue from our true distemper encephalitis cases have infected ferrets and dogs in the same fashion as have strains isolated from other sites in the body and from non-encephalitic cases. Mr. Mansey has stated categorically that vaccine prepared from spleen tissue did not prevent the occurrence of distemper encephalitis in the dog although it lowered the incidence of what he has called the alimentary, pulmonary, cutaneous or ocular forms of the disease. We do not know the evidence upon which he based this statement. We do not recognise these various "forms of distemper" and have seen no evidence whatever to support the contention that immunisation with the products at present available prevents one train of symptoms rather than another. What may have confused the issue is a lack of appreciation of the existence of "hard pad" virus and its tendency to produce encephalitic symptoms. We do, then, on the basis of our present information, deny the existence of neurotropic strains of distemper virus, and

we consider virus obtained from spleen tissue capable of stimulating a complete immunity against distemper infection.

All must have been interested in Mr. Mansey's experiments from which he concluded that mice may be infected with the virus of canine distemper. It has intrigued us much in view of the failure of a number of workers in our laboratories to transmit this virus infection to the mouse by a variety of routes. Mr. Mansey was good enough to acknowledge our provision of a strain of distemper virus for his work, but we did not hear in his remarks proof that the infection of his mice was, in fact, caused by this virus. Workers on virus diseases have, in the past, been misled into thinking they had transmitted an infection to mice, whereas their handling of these animals had stimulated a dormant infection natural to the mouse—even the intracerebral injection of broth has produced this effect. The symptoms which Mr. Mansey described are not inconsistent with the presence of lymphocytic chorio-meningitis virus in his stock mice. This disease was described by Traub in the *Journal of Experimental Medicine*, **63**, 533 (1936).

We would, then, withhold further comment on Mr. Mansey's work until we have had an opportunity to examine it with him, but it must be pointed out that the virus he obtained from the brain of his infected mice has, apparently, not produced distemper when inoculated into dogs and ferrets. We hope that this fact alone will justify our cautious attitude. We cannot, however, leave his contribution without encouraging him to complete his investigation despite the fact that our observations do not support his basic contentions.

The value of Mr. Ian McIntyre's short contribution must have been appreciated. He has used the opportunity of his position in the clinic of the Royal (Dick) Veterinary College to good advantage. Many must have been struck by the low incidence of encephalitic symptoms in his series of cases, and this further enforces our opinion that infection with this virus does not necessarily lead to encephalitis. Why there should be this marked variation is indeed difficult to understand, although there are some who contend that the nature of the disease is changing. He has drawn attention to the pneumonic symptoms to which we probably did not give sufficient prominence in our paper. Dyspnoea is sometimes severe, and congestion and oedema of the lungs is the usual *post-mortem* finding in such cases. In addition, both in these cases and in cases that show no signs of pulmonary involvement during life, we have found, as is stated in the paper, small areas of mainly interstitial consolidation, and we are most interested in his statement that in his cases a similar pneumonia is commonly very extensive. We have seen such cases, but they have not been common in our experience.

It was fortunate that one with Mr. Parry's experience of the nervous states of the dog contributed to the discussion. We accept his contention that in any scientific classification, however tentative, nutritional deficiencies should not be placed under the general heading encephalitis. However, the bulk of the practitioners in this country have used the word encephalitis in a broad and rather loose way. They have applied it to any condition which shows symptoms which might be related to disturbance of the central nervous system, and in face of the difficulty of field diagnosis we have some sympathy with their attitude. We are obliged by Mr. Parry's pointing out a rather serious error in our paper where we accepted lack of vitamin B_1 as a nutritional state which causes convulsions and neurasthenia. Our reading of the literature had led us to believe that some deficiencies of the vitamins of the B group did lead to the dog having convulsions; by accident we related this to vitamin B_1 in constructing our paper. For what it is worth, cases diagnosed by practitioners as encephalitis have responded to the injection of aneurin in large doses.

Mr. Parry appeared to make the point that, in general, poorly nourished animals, and, in some cases, those with a definite vitamin deficiency, were more resistant to virus infection than were the well nourished. The terms "ill nourished" and "well nourished" are hard to define. We still feel that some improper state of nutrition is a factor in allowing "hard pad" virus to produce its full effects. We, too, would agree with Major Pugh's contention that complete rest is conducive to recovery. The difficulty with our dog patients is to give effect to this recommendation without producing in the animal a state of nervous tension through departure from its normal habits.

That Mr. Weipers has contributed to the discussion gives us the opportunity to acknowledge the considerable help we have received from him and from other clinicians. Many have been most patient with us and have appreciated that extensive examinations are often necessary before a good opinion can be expressed. We were pleased to hear his appeal that an adequate case history be provided. Few practitioners appreciate the help and stimulus which the laboratory worker receives when the not very attractive material is covered by the full details of the case. Many of the specimens which we have received during the last three years lost much of their value to us through the practitioner's failure to supply case histories. From the living animal the specimen of choice is a sample of citrated blood, 2 c.c. to 5 c.c. in amount, despatched by post without delay to the laboratories. Where diarrhoea is a prominent symptom a small sample of faeces should also be sent. Tissues selected at *post-mortem* examination are of value, but we prefer to receive the unopened carcase. The most suitable specimens are a small portion of spleen, lung and brain quite fresh, each in a separate sterile container, and other pieces of these tissues and some of the pads in 10 per cent. formal-saline solution. Now that our survey has proceeded so far, we agree with Mr. Weipers that better progress will be made by our having material from outbreaks of disease rather than from odd cases. Mr. Weipers has done us a service in illustrating the difficulties which are associated with research on virus diseases and in emphasising the unstable nature of these agents.

We would not deny the possibility of some antigenic relationship between the virus of distemper and that of "hard pad disease." We would hesitate to accept that the one was just a variant of the other.

Mr. Weipers, too, has remarked on the marked variation in the symptoms of "hard pad disease." Could we expect it to be otherwise when the causal agent seems so uncertain in its effect in our experimental animals? The most interesting condition of the lungs either was not a feature of the earlier cases of the disease or was overlooked. In the latter phases of our investigations we have encountered many cases in which a marked oedema of the lungs was a clinical and *post-mortem* feature of "hard pad disease." We still have difficulty in accepting that the dog which has recovered from an attack of ordinary clinical distemper will not succumb to a natural attack of "hard pad" virus. We have no doubt but that some of the cases of so-called distemper were, in fact, infections with "hard pad" virus, and only in those instances in which it has been proved that true distemper virus was the cause of the illness could we accept evidence on this point.

Mr. Weipers has expressed a cautious view that some protection against naturally occurring "hard pad disease" is afforded by the early administration of ordinary distemper serum. If this is so, the level of protection is indeed low and it may well arise from the fact that many street dogs have been infected with "hard pad" virus and developed some immunity. When such dogs are used for distemper serum production a level of that immunity will be present in the product. Quite recently we have been able to produce anti-"hard pad" serum in reasonably large quantities and the several practitioners who have used it, for both prophylactic and therapeutic purposes, report a value far in excess of anything claimed by the most optimistic for ordinary distemper serum.

Mr. Duckett has evidently recognised a large number of cases of "hard pad disease" and we would agree that the condition takes various forms, such as those on which he has built his classification. We feel that "hard pad" virus produces a general infection and that the progress of the case depends, very largely, on the amount of tissue damage, the symptoms depending on the degree of loss of function in the various organs. We join with him in our appreciation of the difficulty of an early diagnosis and in the suggestion that the sulphonamides be avoided, and it is certainly interesting to learn of the high level of success which Mr. Duckett has attained in his use of liver extract. We have, naturally, concentrated on more specific lines of treatment, and if the earlier reports on the use of anti-"hard pad" serum are substantiated, the use of this product may enable practitioners to effect a satisfactory termination in a much higher proportion of their cases.

Mr. Hewetson has well illustrated the difficulties of the small-animal practitioner in dealing with the class of disease under discussion. We recall the investigation in which we collaborated with Dr. Innes and we hope the latter will find time and opportunity to publish his findings, which we understand would confirm the belief that what we now call "hard pad disease" was present in Britain during the late 1930s. It is remarkable that Mr. Hewetson has only seen hyperkeratosis of the foot pad very recently. Despite his disclaimer, we still incline to the opinion that he, like many of the rest of us, may well have overlooked the pad condition in some of the outbreaks which he has encountered. He has certainly emphasised the wide variation of symptoms which seem characteristic of this virus infection and which are so confusing. He has joined the ranks of those who regard the sulphonamides as of limited usefulness in canine practice. The high incidence of Gram-negative organisms as primary and secondary invaders in diseases of the dog suggests the explanation. He, unlike Mr. Weipers, has found no value in his use of anti-distemper serum, but we will be interested, later, to know whether anti-"hard pad" serum will prove useful in his area. He has asked us directly for our opinion of the forcible feeding of sick animals. We do not have the opportunity to follow this point in a large number of cases, but recently we have used protein hydrolysate to maintain the body condition and to prevent starvation and dehydration in anorexia. Some dogs

will lap this material when they will not take other food and, of course, it can be injected by the intraperitoneal or intravenous route.

It is unfortunate that more time was not available for the discussion of this paper, for we are conscious that there must have been many among the audience who could have made a material contribution to our knowledge of these nervous diseases of the dog. It may be that the somewhat premature publication of our work will stimulate others to contribute to *The Veterinary Record* articles which will add to our picture and help towards a proper understanding of these very baffling conditions.

ABSTRACT

[Renal Tubular Excretion of Penicillin as Inhibited by Coronamide. Beyer, K. H. (1948.) *Amer J. vet. Res.* **9**, 157.]

It is confirmed that the renal clearance of penicillin in dogs is almost equal to the renal plasma flow. It was found that after coronamide (4 carboxyphenylmethane sulphonanilide) the clearance of penicillin decreased to about the glomerular filtration rate or lower.

Coronamide caused no toxic effects when given in four-hourly doses for four months; neither blood nor urine showed chemical abnormalities, nor were changes seen in the kidney at necropsy. Larger doses of penicillin were required to cure experimental infections in dogs and mice when the drug was used alone than when used in conjunction with coronamide. F. A.

REVIEW

[" Ley Farming." By Sir R. George Stapledon & William Davies. Pp. 182 (including index 10 pp.). Faber & Faber Ltd., 24, Russell Square, London, W.C.1. Price 12s. 6d.]

"Ley Farming" is an excellent book covering every phase of grassland production and management. It emphasises the changes wrought by the exigencies of war and the need for maximum production on our farms—the place of grassland as the cornerstone of a rotational cropping policy enabling the maximum cropping consistent with the maintenance of fertility of the soil.

It gives vital statistics concerning the condition of our grassland before the war and since, stressing the vast need for improvement and for an educational policy dealing with improved production of grass swards and the management of those swards.

The advantage of temporary grass as opposed to old permanent pasture—the opportunity it provides of enhancing the stocking properties of the land, the increased nutritional value of the sward produced and the management of leys and re-seeded grassland with this object in view—is well and practically dealt with by those two great authorities Sir R. George Stapledon and Dr. William Davies.

The book is lacking in sections dealing more fully with the management of stock on the swards produced, with the problems which have arisen and with the conservation of the production of grassland for winter food, but as a manual forming the basis of a full and practical knowledge of grassland and grassland management, it is worthy of close study, repeated reference, and a conspicuous place in every veterinary surgeon's library.

N.F.U. AND THE SHORTAGE OF AGRICULTURAL HORSES

The Livestock Committee of the National Farmers' Union consider that an acute shortage of agricultural horses appears likely to occur in the near future. The Council have agreed to draw the attention of breeders to the desirability of continued breeding.

The Livestock Committee also report that it had approached country branches regarding the revised Government policy relating to the export and slaughter of horses. The general opinion was that the imposition of restrictive measures, both as regards slaughter and export, would still further accentuate the decline in breeding, and it was felt that there should be no interference with this plan.

The views of the N.F.U. were recently communicated to the Minister of Agriculture.

THE VETERINARY RECORD
December 4, 1948

DISTEMPER-LIKE DISEASES OF THE DOG

DISTEMPER in the dog has always been a topic which left room for much discussion and difference of opinion. When Laidlaw and Dunkin commenced their work on behalf of the Field Distemper Fund there were many definitions of the disease. When they had completed it there seemed good grounds for a clear conception, based on the demonstration of the causal agent and on a syndrome which was characteristic. However, the difficulty of applying these criteria in practice, especially in the absence of a good alternative diagnosis, led to a rather ready acceptance of most cases of severe catarrhal disease as true distemper. Many clinicians considered that the appearance of nervous complications, particularly chorea, convulsions or paralysis, proved that the previous illness had indeed been distemper.

Laidlaw and Dunkin demonstrated the methods by which good protection might be obtained and since then distemper prophylactics, prepared in accordance with the principles they laid down, have been widely used. The occurrence of " breakdown " in vaccinated dogs has tended to force individuals into one of two camps: either the distemper prophylactics were failing to protect against true distemper, or there was another disease or group of diseases which simulated some clinical aspect of distemper but differed aetiologically. The latter school has gained adherents during the past three years, but there was little good evidence in support until the workers at The Wellcome Research Laboratories declared their conviction that an entity, which they called " hard pad disease," existed and had been confused with the distemper of Laidlaw and Dunkin.

The profession was anxious for a pre-view of this work by MacIntyre, Trevan and Montgomerie and they were importuned to give the paper at Southport which, with its discussion, is published in this number of *The Veterinary Record*. While they dealt with a larger issue and attempted a classification of the nervous diseases of the dog on a micro-pathological and pathogenic basis, their definition of " hard pad disease," tentative as it is, is a material step in the study of the diseases of the dog often included in the generic term distemper. It is hoped that this work will be continued, for it is of great significance to the veterinary profession, and, indeed, to workers on virus diseases.

PHARMACEUTICAL COMMITTEE ON PROFESSIONAL ETHICS

With the object of keeping under review its recently promulgated Statement on Matters of Professional Conduct, the Council of the Pharmaceutical Society has set up an *ad hoc* committee, to be known as the Ethical Committee, consisting of the President, the Vice-President, and six other members of Council. This committee will provide advice in response to requests from members of the Society on ethical questions, and will publish the opinion of the Society on such matters from time to time as required.

* * * * *

DAIRY HERD RATIONS FOR MILK PRODUCTION

The Ministry announces that prospective supplies of protein feeding-stuffs are now such that the ratio of protein to cereals in milk production rations for dairy herds, which was reduced last October, can be restored in and after January, 1949, to the level which was in operation last winter.

As from January, 1949, rations for milk production will be 1 cwt. of protein and 3 cwt. of cereal feeding-stuffs for each 105 gallons of milk sold, or otherwise used for human consumption, in excess of 15 gallons per cow per month, subject to a deduction of 60 lb. of cereal per cow per month. The revised ratio will apply also to milch goats.

R. I. C. HYAM : Foreign Body in the Frontal Sinus

CLINICAL COMMUNICATION

Foreign Body in the Frontal Sinus of a Dog

R. I. C. HYAM, Capt., R.A.V.C.

Subject.—Three-year-old black and tan terrier, whippet cross. Dog.

History.—The dog was presented with a history of a discharging fistula, the exit from which lay in the mid-line of the cranium between the orbital processes. This had first made its appearance three months previously but had apparently healed after ten days. It reappeared about a month later, and despite treatment with hot fomentations and poultices by the owner, had persisted, and when seen some degree of swelling was also noticed.

In all other respects the dog was and had been in perfect health and spirits. On probing it was found that the fistula penetrated to a depth of about one and a half inches in a downward and forward direction. There was some fibrosis of the skin and underlying tissues. An X-ray of the dog's head was made, when a large piece of what was presumed, from the density of the shadow, to be metal, was seen in the frontal sinus and embedded in the bony septum ventrally. There was also resorption and penetration of the frontal bones. These findings are clearly shown on the enclosed X-ray negative.

Operation.—An operation was performed under nembutal anaesthesia, and an irregularly round hole measuring about half an inch in diameter was discovered in the frontal bones giving access to the frontal sinus.

With the aid of a pair of nail clippers, this hole was slightly enlarged anteriorly, when it was found possible to remove the foreign body with a pair of Spencer Wells artery forceps. It proved to be an expended Luger pistol bullet measuring $\frac{1}{2} \times \frac{3}{4}$ inch, split anteriorly as if it had first struck some solid object and ricochetted into the dog's head.

The wound was packed with sulphanilamide and the skin sutured with three Halstead-type sutures. The dog was given a course of Tonophosphan and recovery was uneventful.

The owner, who had been in possession of the dog since it was eight weeks old, was questioned as to when it might have sustained this injury. He stated that the dog had never left his side for more than an hour at a time except on one occasion about nine months previously when it had gone off and not returned for two days—possibly lying unconscious for some of this time. On its return he noticed a small puncture wound in the centre of the dog's head and thought it had been bitten whilst fighting. This wound healed up quickly and without incident, and it was not until the fistula broke through some six months later that the dog showed any further noticeable symptoms of the accident.

This case is remarkable because of the very long time which elapsed before the condition manifested itself and by virtue of the fact that despite the very large size of the foreign body compared with that of the dog, apparently no inconvenience or discomfort, apart from the fistula, which was painless, was experienced by the patient.

ROYAL SANITARY INSTITUTE'S HEALTH CONGRESS, 1949

Next year's Health Congress of the Royal Sanitary Institute will be held at Brighton from May 23rd to 27th, under the Presidency of the Duke of Norfolk.

The section (D) devoted to Veterinary Hygiene will be presided over by Mr. H. T. Matthews, B.V.Sc., F.R.C.V.S., D.V.H., Superintending Inspector, Ministry of Agriculture and Fisheries. Mr. Matthews is also the President of the Sussex Division, N.V.M.A., and we are confident that his colleagues, not only in that Society, but in a nationally wide sphere, will do their utmost to support the section, which meets on May 24th.

* * * * *

Many important changes in the livestock section of the Royal Show, to be held in Shrewsbury from July 5th to 8th next year, were agreed at a recent meeting of the Council. The R.A.S.E. will provide the whole of the class prize money (except breed society special awards and championship trophy prizes). This means that cash contributions for class prizes will not be required from the breed societies. In consideration of the Society's acceptance of this responsibility, the Stock Prizes Sub-Committee is to review the whole of the breed classifications and recommend for the 1950 show and future shows an optimum number of classes for each breed. Special consideration will continue to be given to the demands of breeds which are local to the venue of the Society's Show.

REPORT

The Animal Health Trust

Second Report (September 1st, 1947, to June 30th, 1948)

The expansion of the Trust's interests and activities after the end of the war focused attention upon the name "Veterinary Educational Trust," for it was becoming increasingly evident that many people thought the sole concern of the Trust was the education of would-be veterinarians, comparatively few people being aware of its actual scope. While there was a reluctance to lose, by a change of name, the goodwill which had been built up, it was finally decided to adopt the present title; and events have since proved the decision to have been a wise one.

During the ten months covered by this second report, the Trust completed the acquisition of the land and buildings for the Canine Research Station at Newmarket, and the Farm Livestock Research Station near St. Ives (Hunts.). This means that provision has now been made for research work covering all classes of domestic animals.

It will be noted that all the Trust's Research Stations are within easy reach of Cambridge and, as might be expected, there has been an interchange of ideas between the Trust's scientific staff and their opposite numbers in various departments of the University. It is intended that contact shall be developed with University workers in the fields of veterinary, biological and medical research.

The Research Stations are under the control of Directors, who attend monthly meetings under the chairmanship of the Scientific Director. This system, it is felt, promotes the necessary teamwork while at the same time encouraging individual enterprise on the part of the research staffs. Work has already begun in improvised laboratories at Houghton and Newmarket, but a great many building alterations and conversions have to be carried out before permanent laboratories and clinical accommodation can be made available, and all this requires the granting of permits and the allocation of materials. It is hoped that the Equine Laboratories will be completed by March next year, and the Lanwades Park mansion be converted during the same period into a hostel and central library and museum.

The Trust reports that it is not finding it easy to secure all the expert staff required, but a useful nucleus has been formed; and the policy of employing, at the Research Stations, those who had earlier availed themselves of the facilities for post-graduate studies offered by the Trust in its educational role, will to a great extent solve this problem. The Council is also seriously considering building houses and cottages for the staff on the various estates.

All scientific and other appointments are made to the staff of the Trust as such, and not to a particular research station in the first instance. It is hoped that this will allow of interchange and make collaboration easier; and with the same object in view it is planned to hold informal scientific meetings two or three times a year, in which all may have an opportunity of expressing their ideas.

On the educational side, several students, who could not otherwise have done so, were enabled to complete their courses with the financial assistance of the Trust. A further seven post-graduate scholarships and four research fellowships were awarded during the ten months under review.

Mr. T. K. Ewer, B.V.Sc., DIP. AGRIC. (NEW ZEALAND), was elected to a B.O.C.M. Fellowship so that he could continue nutritional work on the sheep, the work taking place at The Institute of Animal Pathology, Cambridge.

Dr. J. MacLeod was elected to a Cooper Research Fellowship to run from October, 1947, so that he could investigate the complex problem of sheep blowfly. He was stationed at The Veterinary Laboratories, Weybridge, but carried out much of his work in the field in the Solway and the Welshpool areas.

Mr. B. T. Farrelly, M.R.C.V.S., was elected to an Equine Research Fellowship, which began in February, 1948. Mr. Farrelly had been doing pathological work in Dublin but intends to work on the diseases of foals at the Equine Research Station. For the first part of his fellowship he attended a post-graduate course in bacteriology and pathology at the Post-Graduate School of Medicine, London, and subsequently attended a short course in Bacterial Chemistry at Oxford. He is now at Newmarket.

Mr. A. E. Pierce, M.R.C.V.S., D.V.S.M., research officer at the Veterinary Laboratories, Weybridge, has been awarded a Wellcome Veterinary Fellowship from October 1st, 1948. He is proceeding to the United States to continue his studies on trichomoniasis. He will work under Professor B. B. Morgan of Wisconsin University, Madison, an acknowledged expert on this type of disease. Mr.

Pierce will also visit other centres in North America where research into problems of veterinary parasitology is being conducted.

Mr. J. D. Steel, B.V.Sc., a visiting research worker from Australia, is receiving a research grant from the Trust in order to continue his work on the nervous diseases of dogs. Mr. Steel is a lecturer at the Veterinary School, Melbourne, and is over here on study leave.

Colonel John Hickman, M.R.C.V.S., has been given a grant to enable him to go to The Franklyn Institute in the United States of America to study the latest methods in human surgery before joining the staff of the Equine Research Station as an experimental surgeon.

Summarising the Educational awards so far made by the Trust, we have the following figures: Undergraduate Scholarships and Studentships, 38; Final Year Studentships, 24; Post-graduate Research Training Scholarships, 13; Post-graduate Research Fellowships and grants, 18.

Four veterinary surgeons have now been helped to go abroad in order to widen their veterinary experience. Bearing in mind the rapid growth of the Trust from its origin six years ago without funds, these results must be considered both impressive and encouraging.

Other educational work carried out through the Trust includes the making, in co-operation with I.C.I., of several films on certain diseases of dairy cattle and the organisation of lectures on veterinary science. The films so far completed are entitled "Hygiene on the Diary Farm," "Bovine Tuberculosis," "Bovine Mastitis," "Contagious Bovine Abortion," and "Sterility in Dairy Cattle." These films are primarily produced for the farmer, but they have been shown at N.F.U. meetings, Young Farmers' Clubs, and veterinary meetings. They vary in length from nine to 25 minutes showing time, and are available in 35 mm. and 16 mm. copies in black and white sound films.

This year the Trust was fortunate in getting Professor Seekles of Utrecht over to give three lectures on "The Biochemical Approach to Animal Disease."

Clinical, Diagnostic and Hospital Services.—These services are believed to be long overdue by many members of the veterinary profession and the general public. During the year under review the Trust has given much thought to this matter and has held preliminary consultations with the N.V.M.A. It is emphasised that such services would be run with the closest collaboration of veterinary surgeons in general practice.

The report goes on to describe the activities of its various committees: Canine, Bloodstock Industry, Poultry, and Publicity, and the formation of County Committees which have now been established in Berkshire, Cornwall, Glamorgan, Hampshire, Kent and Somerset.

Research

Equine Research Station.—This comprises Balaton Lodge and Lanwades Park. Conversion work at the former has begun, and 56 horses were admitted for observation and treatment in connection with the research programme. At Lanwades Park 68 acres are at present being used as paddocks, and a small pony-breeding unit, consisting of 33 mares (14 Shetland, ten Welsh, four New Forest and five crossbred) and one Shetland stallion, has been established.

Despite the handicaps resulting from failure to obtain licences to make the necessary modifications to the buildings at Balaton Lodge and at Lanwades Park to fit them for research purposes, the second year's work of the Station shows steady progress as regards infertility, parasitology and the diseases of foalhood.

Infertility.—Penicillin treatment of infected mares: Fifteen mares were accepted during the covering season for treatment of genital infections rendering them unfit for service. The infections were in every case caused by a penicillin-sensitive streptococcus, and, in almost every instance, aspiration of air into the vagina was present.

A number of anoestrus mares were accepted at the station during the season and were treated. The stud of Welsh and Shetland mares kept at Lanwades Park was used to investigate sperm survival.

Parasitological Research.—Work proceeded along two main lines during the year: preliminary experiments on the use of sodium fluoride as an anthelmintic for round worms in horses, and the establishment of a method of rearing worm-free foals to be kept on the new paddocks at Lanwades Park and to form a nucleus of parasite-free stock for controlled experiments in the future.

Several horses were also kept for periods varying from a few days up to three months for examination of errors in cardiac function. A steeple-chaser furnished much useful information of a clinical nature, and enabled the Station to acquire a series of what are regarded as valuable electro-cardiographic records of heart block. The second horse, a stallion, provided valuable information upon the dysfunction that may occur as a result of cardiac hypertrophy associated with aortic basal aneurysm.

Canine Research Station.—The acquisition of a 34-acre estate, together with extensive stabling, house, cottage and huts for the purposes of a Canine Research Station, was successfully concluded early in 1948. This estate at Kennett End, adjoining the Equine Research Station of Lanwades Park, offers great scope for development, but little real progress could be made during the year owing to many difficulties, not the least of which was to secure its derequisitioning by the Army. Work has meanwhile been started at Barrow, and has consisted mainly of clinical investigation into conditions such as leukaemia, "night-blindness," and hysteria.

Poultry Research Station.—The Poultry Research Station was established during March, 1948, on the 50-acre parkland adjoining Houghton Grange, which was then available to the Trust.

During the following six months efforts were mainly directed towards the establishment of a control breeding flock and the conversion and equipping of huts to form temporary laboratory buildings.

It is intended to carry out research into diseases of the respiratory tract, such as coryza infections, tracheitis and their differentiation from Newcastle disease, salmonellosis, coccidiosis, blackhead in turkeys and the histology of the urinary and nervous systems.

The report also gives an account of the independent research carried out under Trust auspices, biographical details of new members of the Trust's scientific staff, and an extensive list of committee members. G. P. W.

Trivia

Do You Remember?

Now you old lads of seventy-odd: do you recognise him and can you ever forget him? A figure of about 5 ft. 4 in. in height, borne upon short sturdy legs supporting a body of vast girth, crowned by a round head with cod eyes, fat and flabby cheeks and decorated with a 'tâche which I am sure must have been the model for Bruce Bairnsfather's Old Bill's "walrus." He was invariably to be found, during College hours, in or about the dissecting room or *post-mortem* house. And his drink was beer—what a character he would have made for Charles Dickens. You *must* remember Harry Perry!

I chanced to think of him when pondering over old days, and was reminded of this anecdote concerning the late Sir John M'Fadyean—it went something like this: "Mr. Elphick, did ever I tell you the story of Sir John and the final-year class of stoodents when 'e first cime to the College? We 'ad 'ad a dawg die in 'orspital and it was decided that Sir John and the class would examine it. They cime to the *post-mortem* 'ouse and I 'ad the dawg ready stretched on the tible and they all gathered round. On getting the word I opens 'im up and out pops 'is bowels, all sudden like. Sir John points to a particular piece and says very mysterious: 'Gentlemen, wot is that?' One said it was a impaction; another a telescoping of the bowel, and another a twist. At last Sir John 'e ses: 'Gentlemen, it is the caecum of the dawg, but for Gawd's sike keep it dark!'"

A typical example of Sir John's quiet humour; you can imagine that wonderful twinkle in his eye. H. S. Elphick.

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Lord Mountbatten, who addressed the Royal Empire Society on October 6th, mentioned one of the native states of India—Junagarh—whose ruler, he said, was a qualified veterinary surgeon. This ruler kept some 600 dogs, not in kennels, but in his palace, and he even proclaimed a public holiday in his state to celebrate the wedding of two of the animals. Junagarh was one of the three states—the others being Kashmir and Hyderabad—which held out against the partition, but subsequently, after a plebiscite, it joined up with India.

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A doctor was called in to see a very testy patient.

"Well, sir, what's the matter?" he asked cheerfully.

"That, sir," snapped the sick man, "is for you to find out."

"I see," said the doctor thoughtfully. "Well, if you'll excuse me, I'll go along and fetch a friend of mine—a veterinarian. He's the only chap I know who can make a diagnosis without asking questions."—*Frances M. Block* (*Evening Standard*).

Veterinary Surgeons Act 1948

Reconstituted Council of the Royal College of Veterinary Surgeons

Most of the sections of the Veterinary Surgeons Act, 1948, are now law, and amongst them are those relating to the constitution of the Council of the Royal College of Veterinary Surgeons; in particular, clause 9 of the Act states:

(1) At the first annual general meeting of the Royal College of Veterinary Surgeons held after the commencement of this Act (which will be June 7th, 1949), the existing members of the Council of that College shall vacate office, and as from that meeting the Council shall be reconstituted in accordance with the following provisions of this Act in that behalf.

(2) Subject to the transitional provisions and the provisions relating to Eire hereinafter contained, the Council shall consist of the following persons, that is to say:

(*a*) twenty persons, being members of the Royal College of Veterinary Surgeons, elected in the manner provided by the Charters by members of the College residing outside Eire;

(*b*) four persons appointed by the Privy Council;

(*c*) for each university in the United Kingdom in the case of which an order is for the time being in force under section one of this Act, two persons appointed by that university of whom at least one shall be a member of the Royal College of Veterinary Surgeons.

As sub-section (*c*) of (2) will not come into operation for a little time, the transitional provisions of the Act, under section 12, will operate so far as regards the representation of universities and the present veterinary schools in the United Kingdom. Under these transitional provisions there will be, for the time being, the following representation:

The University of Liverpool, two members; the Universities of Bristol, Cambridge, Edinburgh, Glasgow and London, one member each; The Royal Veterinary College and Hospital of London, the Royal (Dick) Veterinary College, Edinburgh, the Glasgow Veterinary College, one member each.

When the Universities of Bristol, Cambridge and Glasgow establish qualifying courses in veterinary surgery, the Universities will be entitled to two members, one of whom must be a veterinary surgeon. The Glasgow Veterinary College will then cease to be entitled to a member.

When the Royal (Dick) Veterinary College becomes a part of the University of Edinburgh, the University will be entitled to two members, one of whom must be a veterinary surgeon. The Royal (Dick) Veterinary College will then cease to have a member.

When the Royal Veterinary College and Hospital of London becomes a school of the University of London, the University will be entitled to two members, one of whom must be a veterinary surgeon. The Royal Veterinary College will then cease to be entitled to a member.

As it is the first meeting of the reconstituted Council on June 7th next year, the election of members under 9 (2) (*a*) above by members of the College residing outside Eire will have to be for the entire 20 persons who are to be the elected members of Council. In each of the years after next year, of course, the elections will only be for five members each year, as the term of office of a member of Council is fixed at four years.

The Act, under section 11, states that the term of office of the 20 members to be elected next year shall be as follows:

The five who receive the lowest number of votes shall be one year only;

The five who receive the next lowest number of votes shall be two years only;

The five who receive the next lowest number of votes shall be three years only.

The five who receive the highest number of votes shall stay in office for four years.

By this method of members retiring the ordinary electoral procedure can be established.

For the election of members of Council next year, therefore, and for the nomination of members by the various bodies concerned, the Council of the Royal College at the recent quarterly meeting resolved that all nominations should be received by the Registrar at the Royal College of Veterinary Surgeons, 9, Red Lion Square, not later than January 31st, 1949.

Candidates for election to the Council must be members of the Royal College of Veterinary Surgeons who have attained the age of 26 years, or have *bona fide* practised their profession for not less than five years, or been a professor in a veterinary school (Royal Charter of 1892). Each candidate for election must submit a nomination form (to be obtained from the Registrar, 9, Red Lion Square) which must be duly completed by the candidate and signed by the proposer and seconder. This, as has been stated above, must be sent to the Registrar not later than January 31st next.

For the first time voters overseas will receive identical ballot papers with voters in the United Kingdom, containing all the names of candidates for election. The ballot papers will be received by overseas members in sufficient time to enable the completed ballot paper to be sent back so as to reach the Registrar before the final day appointed for their return.

At present Eire is entitled to four representatives on the Council R.C.V.S., which is provided for by the Veterinary Surgeons (Irish Free State Agreement) Act, 1932. With other members of the Council, the Eire members will vacate office at the annual general meeting to be held on June 7th, 1949. The representation of Eire on the reconstituted Council will fall to be determined by agreement between H.M. Government and the Government of Eire, and given effect to by an Order in Council made under section 20 of the Veterinary Surgeons Act, 1948. Such an agreement has not yet been made. The position on and after June 7th next will, therefore, be that if an Order in Council is in force giving effect to an agreement between the two Governments, the number of Eire members appointed or elected to the Council R.C.V.S. will be as determined by the agreement, but if no such Order in Council is in force Eire will not be entitled to representation on the Council R.C.V.S.

Should any member R.C.V.S., eligible for election to Council, desire any information the Registrar will be happy to give any assistance which he can.

INTERNATIONAL CONFERENCE ON HORSE BREEDING

In its current issue *The Horse and Pony News* states that Colonel Lord Digby and Mr. F. T. Day, F.R.C.V.S., were the British representatives at the International Conference held at Utrecht (Netherlands) from September 6th to 8th. They were sent jointly by the British Horse Society and the Royal Agricultural Society of England. Sixty-three delegates participated in the Conference representing the following countries: Austria, Belgium, Denmark, Great Britain, Finland, France, Luxembourg, Netherlands, Norway, Spain and Syria.

"The British representatives contributed very materially to the discussion on the sterility and fecundity problems, and the specimen copies of the Stud Books of the four British Heavy Horse Societies, and the photographs and pamphlets aroused much interest among the other delegates.

"After careful consideration of the position of the horse in relation to the increase of mechanisation in agriculture, the Conference came to the conclusion that the horse still occupied a very important place in the economy of numerous countries, particularly in Europe.

"The Conference also concluded that in order to strengthen the position of the horse industry, every effort should be made to initiate improved methods of production and utilisation, both to make it more profitable and to grade up the horse stock used for draft and any other purpose. Horsebreeding could be made more profitable to-day, and the Conference made recommendations to avoid the present large annual wastage which occurs through infertility.

"They also considered that in order to make the utilisation of horses more profitable it was necessary to improve the methods of management and the types of harness, and particularly the types of carts at present made.

"Various measures were also recommended in order to improve quality and the general level of usefulness of horses."

Weekly Wisdom

"*. . . . Ben Jonson said a good thing: 'Princes,' he said, 'learn no art more truly but the art of horsemanship. The reason is, the brave beast is no flatterer. He will throw a Prince as soon as his groom,' the point being, I take it, that it is important for a Prince to be brought as soon as possible into contact with a living creature who will take no account of his rank . . .*"—Professor Joad (replying to the question: How would you educate a Prince to take his place in a constitutional monarchy? *Sunday Despatch*, November 21st, 1948).

IN PARLIAMENT

The Milk (Special Designations) Bill

FIVE YEAR PLAN TO RAISE STANDARD OF PURITY

In the House of Lords on Tuesday of last week, Lord Walkden, on behalf of the Earl of Huntingdon, Parliamentary Secretary, Minister of Agriculture, presented The Milk (Special Designations) Bill, which, by progressive stages spread over five years, will lead to the prohibition of the sale of milk which is not either tuberculin tested, pasteurised or sterilised.

The Bill proposes to limit the retail sale—or supply—in areas to be specified by the Minister, to the classes of milk which are sold under special designations. Approved milks (*i.e.*, designated milks) in England and Wales will be T.T. (certified) milk; T.T. milk; accredited milk derived from a single herd; pasteurised milk; and sterilised milk. In Scotland the approved milks will be certified milk; T.T. milk; standard milk derived from a single herd; pasteurised milk; and sterilised milk.

The recognition of accredited milk and standard milk as special designated milks will be restricted to a period of five years from the commencement of the Act. The supply of pasteurisation machinery will be a limiting factor, and for this reason it will not be possible to achieve the objects of the Bill in less than five years. When the Bill comes into operation, six months after its enactment, a start will be made by specifying selected urban areas to which it will apply. The Bill will apply to areas to be specified in Orders made by the Minister in consultation with representative organisations. These Orders will be laid before Parliament and will be subject to annulment. The Minister will have power to suspend the operation of an Order in any area in case of need—such as a breakdown of plant—and he may install and operate heat-treatment plant and provide other facilities for that purpose. He may also arrange with local authorities and other persons to provide such facilities in any area in which adequate quantities of heat-treated milk cannot be otherwise provided.

Distributors, selling milk in specified areas, will be licensed, and a revised procedure is laid down for the revocation or suspension of these licences when offences are committed. Enforcement in England and Wales will be by the Food and Drugs Authorities, and in Scotland by local authorities. Penalties will be those prescribed in the Food and Drugs Acts and the Milk and Dairies (Amendment) Act, 1922. The Bill does not apply to Northern Ireland.

The small milk producer-retailer will be most affected by the Bill. He will in due course become liable to install his own pasteurisation plant, or to take his milk to be pasteurised elsewhere before he sells it, or to ensure that he has a certified herd. At present about 30 per cent. of the milk sold in Great Britain is outside the Ministry of Food specification of safe milk. The proportion is much smaller in the urban than the rural areas. In London 95 per cent. of the milk sold is pasteurised. The Ministry of Food estimate that 98 per cent. of the milk distributed to schools is in the "safe" categories.

The conditions for the holding of licences for specified areas are set out, in a schedule to the Bill, in wide terms. They embrace, for example, the examination or testing of animals, the inoculation of animals, the keeping of any animal or herd away from other animals, or other measures for detecting the existence of disease in animals or preventing the contracting or spread of it; the marking, or keeping of records, of animals; the subjection of milk to any heat-treatment process, the cooling of milk, and the conditions in which the milk is kept.

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African Colonies (Tsetse Fly)

On the motion for the adjournment of the House of Commons on Tuesday of last week, Squadron-Leader KINGHORN drew attention to the campaign which has been carried on for some time against the ravages of the tsetse fly in parts of our colonial territories in Africa extending to 4,500,000 square miles, constituting probably the greatest barrier against development in those areas, because it prevented mixed farming from being developed.

Previous Governments obviously had not been unaware of what this terrible insect did, for in 1945, under the Colonial Development and Welfare Act, large grants of money were made in order to combat the sway of the tsetse fly. There was one other instance in East Africa of £275,000, and for West Africa the sum spent, or ear-marked for this work and for research, was £207,000. A further £175,000 was set aside for research into the use of insecticides. It would be of use to hon. Members there, and to the world at large, if the Under-Secretary could give the House some indication of what use had been made of these really big sums of money. He should like to know whether the use of modern insecticides, the success of which they saw towards the end of the war in the use of D.D.T., had suggested a real solution to the tsetse fly problem; and whether full use had been made of all the known resources of modern science which had been put to work on insecticides in the last few years.

Colonial Governments, especially in the lifetime of this Parliament, had shown that they were interested in this matter, and a very interesting report had just been published of a successful experiment in Nigeria, where there had been some real success in wiping out the tsetse fly and other evils from which the natives were suffering. It would be interesting to know whether there had been further activity of that kind. Sir John Boyd Orr had warned us that the world was marching slowly to starvation. Africa, the speaker believed, was going to be a great factor in avoiding that calamity.

Mr. SKEFFINGTON: There were few factors which made such an impression on his mind when he was in Africa recently as the problem of the tsetse fly. It was a very solemn thought that over two-thirds of Uganda and Tanganyika, and one-third of Kenya, were controlled not by the hon. Gentleman but the tsetse fly. He travelled over millions of acres of first-class grassland in Uganda which were denied to cattle because of the tsetse fly.

He would like to put some questions to the Under-Secretary. He was astonished to find that the veterinary and bacteriological services in these three great territories were isolated, and were unaware of the development in each other's domain. There seemed to be no common effort to exchange ideas. He understood that there was a unified service now, but what progress had been made, and was there any arrangement for keeping all the territories fully in touch with development?

He would also like to know if there was any further information about cattle inoculation. In the past attempts had been made to inoculate cattle to prevent them from suffering from the parasite carried by the tsetse, but most farmers decided to run the risk of the fly rather than inoculation, because inoculation certainly killed the animals if the tsetse did not. The use of a drug called "7555" was introduced in April, and he would like to know if it had proved as useful as it was hoped it would be. Had any further provision been made for spraying insecticide from the air?

Mr. E. EDWARD DAVIES: It seemed to him that the most important matter which emerged from the Debate was how far the work had been co-ordinated so that all the Great Powers interested in that problem could have the full benefit of the resources available. We lived cheek by jowl with the Belgians and the French in Africa, and it was very little use our clearing our own area if the problem was being treated differently in another part.

With regard to the cattle in West Africa, would the Minister make some reference to the staffing position? When in West Africa he was impressed with the grave shortage of veterinary surgeons and trained people in control of this branch. What was being done, at a time when there were professional men who could command good salaries in this country, to make conditions attractive in that part of the world so that the best of our good young men could go out without unduly sacrificing their careers and families?

Mr. FOLLICK: He had made a study on the spot of this question. When the Germans were in possession of their colony in East Africa, before the first world war, at Dar-es-Salam, they had one of the finest tropical diseases experimental research laboratories in the world. Since we had taken over that colony that wonderful laboratory had been absolutely neglected. When he was in Dar-es-Salam he went to see the place and it was not used at all—just left to go to pieces. He thought the famous Kleine actually worked at this laboratory.

Between the years 1907 and 1906, in Uganda alone 200,000 people died of sleeping sickness. So that it was a thing that was decimating the population of Africa. He did not think it had yet been found out whether it was the fly, the animal or the man who carried the disease. That was still in process of being investigated. The disease had not only not been got under, it had actually spread, and there were apprehensions at present because it was spreading from Mozambique into Swaziland, and the human and animal population of Swaziland would suffer unless it was held.

The Under-Secretary of State for the Colonies (Mr. REES-

Williams): In reply said that he believed the tsetse fly problem to be African problem No. 1. If we could solve it, we should have gone a long way towards solving the economic, social and, eventually, the political problems of Africa. It affected a vast area, and its solution would change the whole face of Africa and the economy of the world.

"The trouble with tsetse," continued Mr. Rees-Williams, "is that there are so many species of tsetse fly. Some affect cattle and some, human beings. They live on different types of vegetation and feed on different types of animal, so that measures which are effective against one type have no effect upon other types. In British West Africa alone some 10,000,000 people are constantly at the risk of sleeping sickness. The effects upon the cattle population, and consequently upon soil fertility and the balance of farming in Africa, are such as have been mentioned.

"I have been asked by several hon. Members how the problem has been tackled and how we are dealing with it. I hope, so far as I can, to answer them here and now. First of all, certain areas have been cleared and re-settled by various methods. Firstly by selective clearing of the bush, that is, by clearing the type of vegetation on which the tsetse feeds. Secondly, by the use of drugs, both as cures and as prophylactics in human beings and cattle. The drug which the hon. Member for West Lewisham (Mr. Skeffington) mentioned has had an extremely good effect as a cure, and is now undergoing field tests as a prophylactic. We are not quite certain whether it is the complete answer, but there are good possibilities that this is so.

"Thirdly, by spraying with insecticides, although it is too early to form an opinion on the effectiveness of this. Two aircraft have been specially fitted and are now in East Africa, and a helicopter is on order. But sending out large fleets of aircraft or helicopters to Africa is not in itself an answer; the research side, and the scientists who have to study the results of these experiments, must be the deciding factor. It is easier to get aircraft than scientists. Fourthly, there is the control of game, which has been dealt with on a large scale in Southern Rhodesia. We are examining the results of this.

"In addition, we have made available for research since 1945 more than £1,000,000 from the Colonial Development and Welfare Fund. This money is being spent either upon actual research or on development work on the problem of the tsetse fly. The Colonial Governments also have spent a good deal of money on this side of the work. We are also happy in having the assistance of industrial bodies in research. The I.C.I. group and Shell are joining with us in this fight against this noxious insect.

"Finally, there is international collaboration. The hon. Member for Burslem (Mr. A. Edward Davies) asked me a question on this point. There was a conference at Brazzaville this year at which this whole problem was thrashed out with other Powers which have African responsibilities; and in London soon there is to be the first meeting of the International Scientific Committee which will co-ordinate and direct sleeping sickness research in both Europe and Africa. Veterinary surgery is a matter which was also dealt with by the hon. Member for Burslem. We have been very short of veterinary officers, but recently we had a conference with the Registrar of the Royal College of Veterinary Surgeons in London, and also with an official of the Veterinary Association, and we have now got, I think, terms which will appeal to the profession as a whole. I do not think that before, either in status or terms, we were in a position to offer veterinary surgeons satisfactory inducements for them to enter the Colonial Service. The old conditions and terms no longer obtain, however, and we have now got satisfactory conditions for veterinary surgeons in this important and interesting work.

"Here I must pause to mention three reports by scientists which are available to hon. Members. They are those of Professor Buxton, Professor Davey and Dr. Nash. I commend them to the attention of hon. Members in this House, for they will find detailed accounts of the work of the scientists for many years past in these fields. There is also reference to the more humble workers, the small boys who catch the flies. Their work is important because it is the basis of the whole of the scientific work that is done. The standard of measurement of the fly density is known as F.B.H.—flies per boy hour, irrespective of the kind of boy—short, fat, tall or thin, who does the catching.

"To ensure success a combination of all the methods referred to must be and is being used. The key to success is occupation by man of areas cleared by scientific methods and the putting of his cattle on those areas. Several successive stages have to be gone through. First, there is the clearing of the tsetse, then the introduction of water, crops, stocks, cattle, and also the making of communications. Then there is the settlement of people in those areas. Together these steps should produce the answer to the tsetse problem. For example, there is the Anchau resettlement scheme, which has been mentioned. This was made possible by means of a Colonial Development and Welfare grant. There is also a settlement scheme in the Gold Coast and a scheme in Tanganyika called the Shinyanga scheme.

"We must, I think, distinguish between the types of development which can take place in the more fertile and those which can take place in less fertile areas. Where it is quite fertile and people can go and live and farm in the normal way there is no difficulty. The three stages may be put into operation fairly quickly and people settled at a density of not more than 20 to the square mile. In the less fertile areas, however, if there is a density of 20 to the square mile, we are up against a difficulty, because through that infertility there will be a cumulative reduction in the standard of living. For that particular type of country, of which there is a good deal in Africa, and particularly East Africa, there will have to be schemes not of peasant proprietorship, but of large-scale cattle ranching. It will be possible, we hope, to establish in those areas such ranches or co-operative ranches. These will provide much beef for the people of the territory and possibly for an export trade to this country and to other countries in need of beef. We already have a team in Tanganyika investigating this new form of enterprise."

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Questions

Milk and Dairies (1944) Act

Colonel Clarke (November 22nd) asked the Minister of Agriculture when he intends to bring the Food and Drugs (Milk and Dairies) Act, 1944, into operation.

The Parliamentary Secretary to the Ministry of Health (Mr. John Edwards): I have been asked to reply. Delay has been caused by certain legal difficulties which have come to light, but the order to bring the Act into operation will be made as soon as possible. I am sending the hon. Member a copy of a recent circular addressed to local authorities.

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Foxes (Preservation)

Mr. Anthony Greenwood (November 23rd) asked the Minister of Agriculture whether he will inquire into the extent to which foxes are preserved for purposes of hunting; the acreage devoted to artificial coverts for the harbouring of pests; and the practice of constructing artificial earths.

Mr. G. Brown: No, Sir. I have no reason to believe that foxes are preserved to any appreciable extent. Moreover, an inquiry of such a wide range and uncertain character would involve considerable expenditure of time and the results would almost certainly be inconclusive. Without strong evidence to the contrary, I am not satisfied that there is sufficient justification for diverting the pests staffs of C.A.E.C.s from their normal duties for such a purpose.

Mr. Greenwood: Is my hon. Friend aware that the Masters of Foxhounds Association and other fox-hunting interests admit that foxes are preserved for purposes of sport? Is he further aware that leading poultry experts have alleged in poultry newspapers that British housewives are being robbed of millions of eggs because of this practice? Will the Government therefore take a courageous stand to deal with this fox-hunting fifth column on the food front?

Mr. Brown: There may be a good deal to be said for taking a courageous stand, but one has to be careful where one takes the courageous stand. I am not at all aware of the second allegation which my hon. Friend has made, but I would remind him that county executive committees have power under Section 98 of the 1947 Agriculture Act to require the destruction of foxes and there is no evidence that they are not in fact using that power.

Mr. Paget: Is the Minister aware that foxes kill a very great number of rats and rabbits which do far more than equivalent harm?

Mr. Greenwood: Is my hon. Friend aware also that, whatever happens in Northampton, large numbers of poultry have recently been destroyed in my constituency by foxes? Will the Minister therefore try to purge the agricultural executive committees of fox-hunting interests?

No answer was given.

. . . . .

Animal Experiments

Mr. Viant (November 23rd) asked the Secretary of State for

the Home Department how many experiments on cats were performed during the year 1947, under the Cruelty to Animals Act, 1876, with anaesthetics, without anaesthetics, and in which the animals were allowed to recover from the effects of the anaesthetic, respectively; and how many such experiments were performed on dogs.

Mr. Ede: The following table gives the required information:

Number of experiments performed on	Cats	Dogs
(*a*) With anaesthetics	3,888	1,072
(*b*) Without anaesthetics	373	984
(*c*) With anaesthetics from which the animals were allowed to recover (included in (*a*))	296	254

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African Game-Free Zone

Mr. Baldwin (November 24th) asked the Secretary of State for the Colonies what progress has been made with the scheme for the game-free zone along the Tanganyika-Northern Rhodesian border; how many head of wild game have been slaughtered since its inception; and will he give an estimate of the success of the scheme.

Mr. Creech Jones: The game-free area was closed down in May, 1947. A large number of smaller game animals was shot, but I am not in possession of detailed figures. I am advised that the fence, combined with controlled shooting of game and immunisation of cattle, was successful in preventing the spread of rinderpest southwards.

Mr. Baldwin: Will the Minister make other enquiries in regard to this costly experiment, where something like 200 miles of fencing were destroyed and the wild animals were just as numerous after as before the four years' shooting took place?

Mr. Creech Jones: I have been in touch with the local Governments about it, and they declare that the experiment was successful.

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Humane Killers (Firearms Certificates)

Mrs. Manning (November 25th) asked the Secretary of State for the Home Department whether he is aware that a person who wishes to borrow a humane killer to kill an animal elsewhere than in a registered slaughterhouse, has to obtain a firearms certificate from the local chief constable, and whether, in view of the fact that this obligation discourages the use of humane killers in many cases and that there is no danger to human life in the possession of a humane killer, he will consider amending his order on this subject.

Mr. Ede: The Firearms Act, 1937, forbids the use of a humane killer in such circumstances unless it is authorised by a firearm certificate, and I have no power to waive the requirements of the Act. In such a case the certificate would be issued free of charge.

Lieut.-Colonel Sir Thomas Moore: Is the right hon. Gentleman aware that humane killers can be, and have been, used to commit suicide and murder, and is he further aware that inspectors of the R.S.P.C.A. and all the other great animal protection societies are always ready to slaughter animals humanely upon request?

Mr. Ede: I understand that these humane killers, in the hands of careless or ill-disposed persons, can be dangerous to life; and Parliament has imposed this restriction on their use.

Mr. E. P. Smith: Do these humane killers have to be surrendered to the police after use, and, if so, how does the right hon. Gentleman's Department dispose of them?

Mr. Ede: No, Sir. The humane killer is subject to particular Acts of Parliament, one of which, I believe, was introduced by the hon. and gallant Member for Ayr Burghs (Sir. T. Moore) when he demonstrated to the House how it could be used by putting it to his forehead, but, unfortunately, he missed.

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Knackers' Yards (Licences)

Squadron-Leader Fleming (November 25th) asked the Minister of Health what steps have been taken to ensure that every knacker's yard is licensed by the local authority concerned.

Mr. Bevan: I have no reason to think that the Food and Drugs Act is not being enforced. But if the hon. and gallant Member has information otherwise, perhaps he will let me have it to investigate.

Therapeutic Substances (Animals)

Mr. Viant (November 25th) asked the Minister of Health what happens to the horses, cows and sheep after they have been used for the manufacture and production of vaccines, sera, anti-toxins and similar materials, at the various premises licensed by his Department under the powers of the Therapeutic Substances Act, 1925; and, when the carcases are sold for food purposes, whether animal or human, what steps are taken to secure that they are free from disease and are otherwise fit for use as food.

Mr. Bevan: My own powers extend only to the standards of the therapeutic products in these cases.

NOTES AND NEWS

Diary of Events

Dec. 4th.—Meeting of the Biochemical Society, in the Middlesex Hospital Medical School, London, W.1, 11 a.m.

Dec. 6th.—Meeting of the Farmers' Club, at the Royal Empire Society, W.C.2, 2.30 p.m. (Mr. A. B. Buchanan-Smith, C.B.E., D.SC.: "Inheritance of Milking Capacity in Dairy Cattle.")

Dec. 7th.—N.V.M.A. Small-Animals Sub-committee, at 36, Gordon Square, W.C.1, 2 p.m.

Dec. 8th.—N.V.M.A. Technical Development Committee, at 36, Gordon Square, W.C.1, 11 a.m.

Dec. 9th.—Meeting of West of Scotland Division, N.V.M.A., Clinical Club, at the Glasgow Veterinary College, 7.30 p.m.

Dec. 15th.—N.V.M.A. Editorial Executive Sub-committee, at 36, Gordon Square, 2.30 p.m.

Dec. 15th.—Meeting of the Section of Comparative Medicine, Royal Society of Medicine, at 1, Wimpole Street, W.1, 5 p.m.

Dec. 17th.—Meeting of the Association of Veterinary Teachers and Research Workers (Northern Region), at Runcorn (Evans' Biological Laboratories), 11 a.m.

Jan. 5th, 6th and 7th, 1949.—Quarterly Meetings of Council and Committees, N.V.M.A., in London.

Jan. 10th to 13th, 1949.—R.C.V.S. Council and Committees, 10, Red Lion Square, London, W.C.1.

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N.V.M.A. Meetings in London

In order that they may have the opportunity of reserving accommodation, the attention of those concerned is drawn to the fact that the next Quarterly Meetings of the Council and Committees of the Association will be held in London on January 5th, 6th and 7th, 1949.

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Adviser on Animal Health to the Secretary of State for the Colonies

The Overseas Services Committee of the N.V.M.A. took advantage of the occasion of the retirement of Mr. John Smith, O.B.E., M.R.C.V.S., D.V.H., and his succession by Mr. Robert Simmons, C.B.E., M.R.C.V.S., to have a small informal luncheon party at the Russell Hotel, London, W.C.1, on Monday, November 29th. Sir Charles Jeffries, K.C.M.G., O.B.E., Deputy Under-Secretary of State in the Colonial Office, was also a guest. Appreciation of Mr. Smith's work was expressed by Dr. Carmichael, Chairman of the Committee, who pointed out that Mr. Smith had pioneered a development greatly to the advantage of animal health in the Colonies. In welcoming Mr. Simmons as his successor and wishing him well in his appointment, the Overseas Services Committee are conscious that they have a colleague who will continue the high level of co-operation and goodwill which has been established.

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Children of Danish Veterinary Surgeons

The Association would very much like this year to return the hospitality shown to the children of British veterinary surgeons by members of the Danish Veterinary Association and their families if suitable arrangements can be made, and therefore propose to extend to these children, through Dr. Plum (the President), a very cordial invitation to come to England next summer.

Members who would like to join in this scheme are asked to notify the General Secretary as soon as possible, *and not later than December 10th*, in order that arrangements may be started at once. It is a little difficult to give an idea of the time convenient for all concerned because it is believed that Danish school holidays do not quite coincide with holidays in this country, but late July and early August may possibly fit in.

PERSONAL

Professor Worden's Animal Health Trust Appointment

The Animal Health Trust announces that Professor A. N. Worden, M.A., B.SC., M.R.C.V.S., A.R.I.C., Milford Professor and Director of Research in Animal Health, University College of Wales, Aberystwyth, has been appointed biochemist to the Trust's Farm Livestock Research Station at St. Ives, in Huntingdonshire. Professor Worden will be taking up his duties in the New Year, and will supervise, in collaboration with the Scientific Director and the Directors of the various Research Stations, the development of biochemical and nutritional programmes of the Trust.

Professor Worden is 32. He was educated at Queen Elizabeth's School, Barnet, The Royal Veterinary College, London, and St. John's College, Cambridge. From 1938-41 he was a research student in animal health of the Ministry of Agriculture and Fisheries at the Lister Institute of Preventive Medicine, where he worked in the Division of Nutrition under Dr. Harriette Chick. He then worked at the Veterinary Laboratory, Weybridge, and, for a short time, at the Rowett Research Institute, in charge of experiments on dairy cattle.

From 1942-45, Professor Worden was a member of the staff of the Institute of Animal Pathology, collaborating with Professor F. Blakemore, M.R.C.V.S., D.V.S.M., and with workers at the Dunn Nutritional Laboratory on nutritional biochemical problems.

In March, 1945, he was appointed First Milford Professor and Director of Research in Animal Health, University College of Wales, Aberystwyth, where he built up a research department numbering about 20 graduate staff and research students for the study of problems on animal health and production, with special reference to nutritional factors and to the pasture and animal relationships.

Since 1941 he has been Honorary Secretary to the Institute of Animal Behaviour, and Scientific Secretary to the Universities Federation of Animal Welfare, on behalf of which he has edited a text-book entitled "Care and Management of Laboratory Animals." In addition to various scientific societies and committees, he is a Member of the Council of Agriculture for Wales, and was for two years a member of the Carmarthenshire Agricultural Executive Committee.

Professor Worden is particularly anxious to link studies into the causation and prevention of disease with those concerned with veterinary metabolic disorders. As soon as the Livestock Station can be started, nutritional problems of the adult animal, such as bloat, and the many troubles arising from incorrect feeding of the young growing animal will be investigated.

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Professor L. de Blieck

Professor L. de Blieck, Director of the Veterinary Faculty, Institute of Tropical Hygiene, Utrecht, spent a few days in England last week, to visit Weybridge and farms where poultry research is in progress. As Chairman of the General Purposes Committee, he also discussed arrangements for the International Veterinary Congress to be held in London in 1949.

The Iodine Educational Bureau marked the occasion by a luncheon at which the guests included Professor de Blieck, Sir Daniel Cabot, Professor Dalling, Dr. R. F. Montgomerie (President, N.V.M.A.), Professor G. H. Wooldridge, Dr. W. R. Wooldridge, Dr. W. S. Gordon, Dr. H. H. Green, Mr. H. R. Heywood, Dr. C. S. Hopkirk and Dr. S. H. Whitworth.

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Births.—Herbert.—On Sunday, November 21st, 1948, at the County Maternity Home, Aberystwyth, to Magarette (*née* Rees), wife of T. G. G. Herbert, M.R.C.V.S., Kingsway, Aberaeron, a son—David Rhodri ap Gruffydd.

Rowe.—On November 15th, 1948, at Berkly Hospital, to Ruth (*née* Grace) and J. F. W. Rowe, M.R.C.V.S., Coombe Mount, Wotton-under-Edge, a daughter—Helen.

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Marriages.—Halpin—Milne.—On September 11th, 1948, at The Church of Our Lady and St. George, London, E.17, by the Right Rev. Mgr. W. O'Grady, V.G., Francis Brendan Halpin, of Eltham, S.E.9, to Vivienne Milne, of Leyton, London, E.10.

Renwick—Maclean.—At Alor Star, Kedah, Federation of Malaya, on November 12th, 1948. Charles Campbell, M.R.C.V.S., to Mary I. G. Maclean, daughter of the Rev. Mr. and Mrs. Maclean, Edinburgh. The groom is State Veterinary Surgeon Kedah (Malayan Veterinary Service). Veterinary surgeons present were Messrs. R. A. B. Stanhope, John Davie and A. H. Foster.

The Yorkshire's Lady President: Erratum.—In announcing, in our last issue, the election of Miss Christine A. Woods, M.R.C.V.S., as President of the Yorkshire Veterinary Medical Society, we expressed our belief that this was the first occasion upon which a Division of the N.V.M.A. had bestowed this honour upon one of its lady members. While Miss Woods is the first lady recipient of this honour at the hands of the "Yorkshire," we apologise to her and the Society, also to Mrs. M. L. Harris and the Sussex Veterinary Society, for overlooking the fact that this "pioneer" distinction was conferred upon Mrs. Harris by that Division for the year 1942-43.

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R.C.V.S. OBITUARY

Thompson, Arthur Joseph, Lieut.-Colonel, R.A.V.C. (Retd.). Graduated London, December 17th, 1902. Died at Auckland, New Zealand, July 3rd, 1948.

The Late Major J. N. Glass, M.R.C.V.S.

In a tribute to Major J. N. Glass, M.R.C.V.S., whose death we recorded with deep regret in our last issue, the *Melton Mowbray Times* refers to his 40 years work as a veterinary surgeon in the town and district and an acknowledged expert as a judge of horses which made his opinion valued all over the country and abroad, and brought him a wide and varied circle of friends, to whom his passing has come as a severe loss.

Major Glass "vetted" for the Duke of Windsor and the Duke of Gloucester when they had stables in Melton, and he was called into consultation both here and in Ireland when clients were contemplating buying hunters. He had hunted with the Quorn, the Belvoir and the Cottesmore; he lectured to the Quorn and Belvoir Pony Clubs; and he often gave his services at shows and hunter trials. Major Glass's last task was the honorary one of attending the Belvoir hunter trials at Hose. He drove himself back to Melton that day and was taken ill the same night.

He had other sporting interests, among them tennis, golf and shooting.

In the 1914-18 war he served in the R.A.V.C. and was in Gallipoli with Sir Waldron Smithers. He was mentioned in despatches and rose to the rank of major.

At the funeral service, at Melton Parish Church, the vicar, the Rev. C. M. S. Clarke, said that Mr. Glass belonged to a Melton which was hardly known to-day. He was a man who lived well, who knew all sorts of the community and was held in respect by all. He did his work conscientiously and well, and such a man must have had a tender heart with compassion both for the animal world and for humanity in general. Of all that was good and useful and jovial in life he was a true representative and his friends paid tribute to him and his work.

There were many Hunt representatives at the service, the large company also including Mr. George Gibson, M.R.C.V.S., of Oakham.

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AN IMPRESSION OF THE N.V.M.A. GRASSLAND CONFERENCE

Writing in *The Fertiliser and Feeding Stuffs Journal* for November 17th, Mr. F. E. Corrie, B.SC., N.D.A., N.D.D., said:—

"I attended this conference on both days and I should like to add my congratulations to the many which must have been received by the Association on the arrangements and on the intense interest of the subjects chosen for papers and subsequent discussion. It was remarkable that the readers of the papers were able to compress so much information into the short time allowed them. . . . Having heard all the papers, I was unable, as a confessed mineral enthusiast, to avoid the impression that somehow the agronomists had in the past failed the veterinarians.

"The speakers on the first day told us a great deal that was not new to us and, in my opinion, they failed to give the veterinarian even a guide to the mineral composition of pastures, a fact the more remarkable because of the nature of the papers read on the second day.

"We are, of course, very ignorant of the mineral composition of British grassland. We have now, however, an opportunity to remedy this deficiency in our knowledge. The National Agricultural Advisory Service is well equipped with analytical chemists, and I know that at least in some areas these chemists are analysing samples of local grass and grassland products and, in many cases, are investigating mineral content, including trace-element status. There would appear to be no reason why this kind of work should not be extended to commence a proper survey of the composition (including the mineral status) of British grassland.

"I may be wrong, and if so I apologise now, but I do not think there is any machinery in existence which automatically passes on

the data collected by these chemists of the N.A.A.S. to the biochemists and other research workers in the veterinary field who are able properly to interpret the data, and to whom such information would be of incalculable value, if only as a guide.

"I was so impressed by the success of the conference that I do earnestly hope that something more may come of it than has come of many conferences in the past. There has been far too much hiding of information, often of considerable importance, in local files and offices. If only in the future we could be sure of the pooling of such information and especially of the direction to it of those engaged in research associated with it, we should make some real progress."

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EFFECTS OF OESTROGENS ON MILK SECRETION

Close upon the work recounted at the recent Grassland Conference of the N.V.M.A. by Dr. D. H. Curnow, upon the discovery of potent oestrogens in growing clover in certain areas of Western Australia, affecting deleteriously the local sheep-rearing economy, through setting up changes in the reproductive organs of both sexes, comes a preliminary announcement (*Nature*, November 27th, 1948, p. 845) from a group of workers employed at the National Institute for Research in Dairying, Shinfield, near Reading (Bartlett, S., Folley, S. J., and Rowland, S. J.) and the Courtauld Institute of Biochemistry, Middlesex Hospital, London, W.1 (Curnow, D. H., and Simpson, Sylvia A.) which, they say, is in harmony with the theory that the galactopoietic effects of spring grass in lactating cows may be due to the presence of oestrogen.

There is, they state, a progressive decline in the non-fatty solids of cows' milk during the winter months followed by an immediate and substantial rise on turning the cows out to grass in the spring. It is, of course, widely known that cows on going out to grass often show an increase in the milk yield. Attempts to simulate these galactopoietic effects by feeding liberal supplements of carbohydrate or protein to the ordinary winter rations gave no response. In speculating upon the nature of the supposed galactopoietic factor in spring grass the recent Australian work mentioned above comes to mind, as well as that of Folley and his associates which had shown that the administration of oestrogens will increase the solids content of cows' milk.

Samples of herbage were cut from a number of pastures during the period of active growth and, after suitable extraction, tested for their oestrogenic activity by the effect on the weight of the uteri of immature mice, the extracts, dissolved in arachis oil, being dosed in six subcutaneous injections over three days, with autopsy 18 hours after the last injection. Extracts thus obtained, in a first series, from two samples cut in the early flowering stage, in May, 1948, gave significant uterine weight increases over the (untreated) controls; while, in a second series, extracts from a number of pastures at the second seasonal cutting, on June 23rd, 1948, cut in the pre-flowering stage showed probable increases in the case of the two grass samples tested and an undoubted increase in the case of the clover sample also tested, whereas a hay sample was negative, producing no significant increase in uterine weight as compared with the controls.

These results, according to the authors, justify further work.

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ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

FOOT-AND-MOUTH DISEASE:

Hants.—Bridge Farm, Hare Lane, Ashley, New Milton, Hants (November 22nd); Ashley Manor Farm, King's Somborne, Stockbridge, Hants. (November 23rd).

Norfolk.—No. 1 Council Smallholding, Kirby Bedon, Norwich, Norfolk (November 23rd); Grange Farm, Bramerton, Norwich, Norfolk (November 24th).

Wilts.—Manor Farm, Chicklade, Tisbury, Wilts (November 23rd).

SWINE FEVER:

Devon.—Ministry of Food Slaughterhouse, Crown and Anchor Way, Paignton, Devon (Premises of origin: S. H. Steven, 2, Hillside, South Brent, Devon) (November 25th).

SHEEP SCAB:

Lancashire.—Black Cowm Farm and Cockhall Farm, Whitworth, Rochdale, Lancs.; Cowm Top Farm, Shawforth, Rochdale, Lancs.; Scout Barn Farm, Turn, Ramsbottom, Manchester (November 22nd); Sky House Farm, Edenfield, Ramsbottom, Manchester (November 23rd).

Yorkshire (W.R.).—Ramsden Farm, Walsden, Todmorden, Lancashire (November 22nd).

R.C.V.S. Graduates and Practice in Canada

PROSPECTS AND REQUIREMENTS

The Royal College has forwarded to us, for the information of any of our readers who may be interested therein, an official statement concerning the prospects of members of the profession in Great Britain securing positions in Canada and the requirements for entering the profession there, which the Superintendent, European Emigration for Canada, was prompted to send in consequence of the receipt of many enquiries. In his covering letter the Superintendent stresses that the utmost difficulty is met in endeavouring to pre-arrange employment before arrival in Canada, as employers attach considerable importance to the personal interview in addition to satisfying themselves as to the experience and qualifications of the applicant. The addresses of the Executive and Professional Offices of the National Employment Services are shown for the benefit of those who wish to make use of these offices, following their arrival in Canada: "no useful purpose is served by communicating with these offices beforehand."

The statement is as follows:—

1. At the present time in Canada there is a shortage of veterinarians. However, in a year or so the situation may well be materially different. Canada's two veterinary colleges are filled to capacity, owing to the large number of veterans attending. Commencing next year (1949), these expanded classes will be graduating and providing double the normal annual supply for a matter of four or five years. After this period, there is no reason to doubt that a sufficient number of veterinarians will be graduating to take care of normal needs for the country.

2. The Canadian Veterinary Medical Association is national in character and represents all veterinarians in Canada. Notwithstanding, each province controls the practice of veterinary medicine within the province by Acts of the Provincial Legislature and each provincial association maintains its own practice board and regulates the practice of veterinary medicine within the province.

3. To be eligible for membership in the Canadian Veterinary Medical Association, a veterinarian must first be registered in at least one of the provincial associations to practise within that province and must remain in good standing within the provincial association.

4. In general, a veterinarian entering this country from any other country and desiring to practise his profession in Canada must first produce to the provincial association concerned his graduation credentials. If it is found that he has graduated from a college or university equal in standing to our Canadian institutions, then the practice board will set an examination which the applicant will be required to pass before being registered to practise veterinary medicine. In the province of Quebec the Act requires that a veterinarian must have been a resident of Canada for a period of at least five years before being eligible for registration to practise in that province.

5. Veterinarians entering Canada from any other country and desiring to practise their profession in Canada are advised to first communicate with the secretary of the provincial association of the province in which they desire to locate for the purpose of obtaining full particulars regarding the practice law of that province.

6. The majority of veterinarians in Canada are not in salaried positions but conduct their own practices. However, there are certain opportunities for employment on a salary basis with provincial and municipal governments. With regard to positions with the Dominion Government, newcomers to Canada would not be eligible because one of the qualifications required by the Civil Service Commission is five years' residence in Canada.

7. In Canada the selling of practices is not prevalent and, therefore, there would be little, if any, opportunity of purchasing a practice.

8. With regard to salaries in the few salaried positions which are open, it is difficult to give any definite figures because these vary greatly according to the locality and the type of work required. However, as an indication, it may be mentioned that, in the Dominion Government Service, salaries range from $2,700 per year to $4,800 per year. For part time service, the salary rate is $15 per day for incidental services.

9. The association fees for the different provincial associations vary at present from $6 to $10 per year. It is believed that these fees will be increased in many instances during the coming year.

10. The names and addresses of the secretaries of the different provincial associations from whom detailed information may be obtained are as follows:—

Dr. E. S. Notting, Secretary, Prince Edward Island Veterinary Association, Jordan Building, Queen Street, Charlottetown, P.E.I.; Dr. L. S. Doyle, Registrar, New Brunswick Veterinary Association, Moncton, N.B.; Dr. E. E. Ballantyne, Registrar, Nova Scotia Veterinary Association, Truro, N.S.; Dr. P. Villeneuve, Secretary-Treasurer, Quebec College of Veterinary Surgeons, 2018 Jeanne Mance Street, Apartment 4, Montreal, P.Q.; Dr. G. A. Edge, Secretary-Treasurer and Registrar, Ontario Veterinary Association, Box 37,

Postal Station "F", Toronto, Ontario; Dr. J. M. Isa, Secretary-Treasurer and Registrar, Manitoba Veterinary Association, Veterinary Laboratory, University of Manitoba, Fort Garry, Manitoba; Dr. A. Chambers, Secretary-Treasurer, Saskatchewan Veterinary Association, 277, Angus Crescent Regina, Sask.; Dr. J. C. Wainwright, Secretary-Treasurer, Alberta Veterinary Association, 26, Central Building, Calgary, Alberta; Dr. F. W. B. Smith, Secretary, British Columbia Veterinary Association, 850, Hastings Street West, Vancouver, B.C.

11. This material has been concurred in by the Canadian Veterinary Medical Association.

12. It is suggested that any applicant qualified in the above profession should register at the nearest executive and professional office of the National Employment Service on arrival in Canada, the addresses of which are:—

Maritime Region—Moncton, N.B.: Dominion Government Building, Phone 9105; Quebec Region—Montreal, Quebec: 10, St. James Street E., Phone MA 9101; Ontario Region—Toronto, Ontario: 200, Bay Street, Phone AD 8341; Prairie Region—Winnipeg, Manitoba: 395, Main Street, Phone 98-171; Pacific Region—Vancouver, B.C.: 475, Howe Street, Phone PA 7321.

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A New Mycobacterial Infection in Man*

Veterinary workers have long been sorely perplexed with a skin disease in cattle to which it has been impossible to assign a precise nomenclature. Their perplexity has arisen mainly because the disease when it occurs may produce reactions in the course of tuberculin testing which point to a tuberculous infection, which, nevertheless, as ordinarily observed, is not confirmed *post mortem*. Acid-fast bacilli can usually be detected in the skin lesions, but these bacilli have neither been cultivated on artificial media nor transmitted experimentally to the usual small laboratory animals used to determine the pathogenicity of tubercle bacilli of mammalian or avian type.

An interesting investigation into a skin disease among six human patients in two mixed farming districts (Bairnsdale and Colac) in Victoria, Australia, has come to notice which may possibly provide some indications for the further elucidation of the bovine disease and which is, in any case, a valuable contribution to the comparative study of the diseases set up by the genus *Mycobacterium*. The patients varied from 2½ to 51 years of age, the first case coming under notice in 1940 in the form of a biopsy specimen from the margin of an ulcer in the skin of the leg of a boy when the tissues were found to be teeming with acid-fast bacilli. In all cases, ulceration took place characterised by indolent extension from the centre of a small breach in a solitary area of inconspicuous, slightly irritable induration. The ulcers were stubbornly intractable to ordinary methods of treatment. The lesions were complicated by pyogenic reaction and oedema. Often the therapeutic measures taken led to acceleration and aggravation of the condition. Sloughing and denudation of large areas took place with little or no disturbance in health in the absence of complications. Spread was by extension, often eccentric, of marginal induration, with breakdown resulting in steep-sided or even undercut edges of scalloped outline exposing necrotic-looking tissue on floor and walls. Occasionally the subcutaneous spread resulted in nodules of infiltration and even areas of ulceration beyond the margin of the main lesion. Apparent healing at one part of the margin might accompany focal necrosis with congestion at another. After separation of the sloughs and as the ulcer extended, an abundant gelatinous mass, like blubbery granulation tissue, developed from the exposed fascia.

Histologically, the tissue outlining the ulcer wall and floor was necrotic, necrosis being most extensive in the fatty tissue, in which it extended beneath the dermis for varied distances. In and about these areas tenaciously acid-fast bacilli were found in enormous numbers, in every instance grouped characteristically in sharply defined oval or rounded masses often as great in diameter as fat cells, as well as in smaller groups and scattered units. The bacilli seemed to have become enclosed within distended phagocytes, though nuclei often could not be seen. The blood-vascular engorgement, oedema and polymorph emigration about the necrotic focus appeared to be correlated with the degree of pyogenic infection. In the indolent phase the cellular accumulation was mainly of macrophages, together with scattered lymphocytes and plasma cells. No sign of tubercle follicles, giant cells, endothelioid grouping or caseation was seen. Though the filling of the phagocytes in the subcutaneous tissue with acid-fast bacilli recalled the macrophage reaction seen in this situation in leprosy, there was nothing clearly pointing to that disease to be observed in the lesions.

Similar lesions had been described by Cilento (1942) in Queensland, in which acid-fast bacilli not identifiable culturally or experimentally as tubercle bacilli were found in the depth of clinically indolent and intractable tiny single ulcers.

Experimentally, the Victoria condition was investigated using material derived from three patients upon 76 white rats, 16 white mice, 12 guinea-pigs, two rabbits, one fowl and three lizards. Intraperitoneal inoculation of male rats with either human material or material obtained from a previously infected rat led to ascites invariably, the first sign of disease being swelling of the scrotum with fixation of the testes. This was evident at any time between two and nine months, and usually four or five months, after inoculation. Swelling of the abdomen varied, the rat becoming sometimes enormously distended two weeks after swelling of the scrotum was first observed, or the swelling became gradually increased during a period of three months. Gross subcutaneous oedema was unusual. Sometimes the rats died without any outward sign of disease other than a swollen abdomen and scrotum. Otherwise, if they survived this stage of the disease, ulceration of the scrotum occurred sooner or later, commencing as a small red area which rapidly developed a hard black crust. Subsequently, perforation of the scrotal wall occurred, with escape of the fluid. In some cases ulceration was so extensive as to expose the testes. Healing of the ulcer now proceeded. After a month or two oedema of the tail or feet and rarely of the face was observed and was followed by ulceration. The duration of the disease from the time of inoculation until death was about 12 months. During the last weeks of life, the rats appeared sick and miserable, but before this, in spite of having suffered from cutaneous oedema and ulceration for many months, they usually remained in good general condition with bright eyes and healthy coats. In rats with ascites the volume of fluid varied from 0·5 to 200 ml., and it was yellow and turbid and clotted readily. It contained many inflammatory cells, chiefly large mononuclear phagocytic cells, many of which contained acid-fast bacilli varying from one or two to a mass of hundreds filling a cell. The epididymis commonly showed at one or both ends conspicuous white lesions 0·5 to 5 mm. in diameter and whitish lesions were also present on the adjacent parts of the peritoneal surface of the scrotum. There were often patches of white fibrinous deposit on the surface of the peritoneal organs and minute grain-like lesions were occasionally seen in the omentum. *Post-mortem* examination of rats in which the scrotal wall had been ulcerated and perforated showed no macroscopic peritoneal lesions. In the thorax a small amount of yellow fluid was sometimes found, and this contained cells but usually no organisms. When subcutaneous oedema was present the fluid in the tissues was usually clear and colourless and contained only very occasional cells and bacilli. The ulcers of the oedematous tail and feet varied in depth and contained on the surface pyogenic bacteria as well as acid-fast bacilli. In the lungs no lesions associated with acid-fast bacilli were encountered and microscopical examination revealed a few acid-fast bacilli on one occasion only.

Female rats inoculated intraperitoneally never showed swelling of the abdomen, but oedema of the limbs or tail occurred in some, accompanied by ulceration. One female, inoculated intracerebrally and killed after eight months, showed typical ulceration of the tail and a hind foot. After subcutaneous inoculation about one half of the rats developed no lesions, while in the case of the others an ulcer developed at the site of inoculation after intervals varying from seven to 20 months, but apart from these local ulcers no macroscopic lesions were found *post mortem*.

In mice the infection with acid-fast bacilli was established by inoculation of either human material or infected fluid from rats or other mice. These mice were all males. Whereas in rats gross oedema of the subcutaneous tissues was rare, in mice it was common and marked and involved the whole of the body and head so that they were two or three times their normal size and presented a curious appearance, although just before death they were bright and lively. The mice usually died in four to six months, the period between the first sign of swelling and death ranging from ten to 40 days.

Guinea-pigs failed to show any lesions of the above kind even after observation extending to 15 months. One male rabbit died after intraperitoneal inoculation in 15 months, the only lesions being yellowish areas on each epididymis, smears of which were teeming with acid-fast bacilli. Similar lesions confined to the epididymis were observed in another male rabbit dying ten months after intravenous and intraperitoneal inoculation with ascitic fluid from an infected rat. No macroscopic lesions developed in a fowl killed 13 months after intraperitoneal inoculation. No infection could be set up after intraperitoneal inoculation in three blue-tongued lizards.

* A New Mycobacterial Infection in Man. I.—Clinical Aspects (MacCallum, P.); II.—Experimental Investigation in Laboratory Animals (Tolhurst, J. C., & Buckle, G); III.—Pathology of the Experimental Lesions in the Rat (Sissons, H. A.); IV.—Cultivation of the New Mycobacterium (Buckle, G., & Tolhurst, J. C.). *J. Path. and Bact.* (1948.) **60**. 93-122. With 18 text-figs.

Efforts to cultivate the acid-fast bacillus from human patients or infected rats having failed, it was assumed that the organism needed some special factor for growth, but all attempts to cultivate the bacillus on a variety of media with such additional requirements in view failed when the temperature at which the media were kept was 37° C. or above 37° C. Eventually growths were obtained on yolk agar medium but the results were irregular and could not be confirmed. At this point the fact that the lesions in man were on the skin and those in rats in the scrotum or on the tail and limbs suggested that the organism might require a temperature below 37° C. It was then realised that the successful cultures previously obtained had been incubated in an apparatus with an unsatisfactory heat circulation and that return to the usual incubators had coincided with the cessation of positive results. Cultures made from a number of stored fluids placed in an incubator adjusted to 33° C. then disclosed growth from several. Though growth was at best slower and scantier than that of human tubercle bacilli, no further difficulties were encountered. Growth was found to be satisfactory at 30° and 33° C., poor at 25° and 37° C., and absent at room temperature (17° to 23° C.) and at 41° C. On suitable media primary growth was usually visible in the fourth week at 30° or 33° C. The most suitable media were yolk agar (one part of egg yolk to three parts of sterile 1·25 per cent. agar dissolved in saline to which was added 1 per cent. of a 2 per cent. solution of malachite green) and Petragnani's medium. Low concentrations (1 per cent. and 5 per cent.) of glycerine enhanced growth. The mature colonies on Petragnani were usually pale lemon or lemon, occasionally resembling that of dry mustard. On yolk agar there was little or no pigment production. Cultures produced the typical ascites in male rats three to five months after intraperitoneal inoculation.

It is concluded that the causal organism is distinct from any of the hitherto described mycobacteria. Whatever may turn out to be its relationship with the mycobacteria causing obscure disease in the domesticated animals, the Melbourne workers have certainly contributed valuable pointers to further investigation in so far as the organism studied by them has shown a very unusual range of pathogenicity on experimental inoculation into the smaller laboratory animals and what, but at first sight only, seemed rather odd temperature requirements for successful cultivation on artificial media.

J. T. E.

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INTERNATIONAL HORSE SHOW, 1949

Subject to the approval of the F.E.I. (states the *Horse and Pony News*), the 1949 International Horse Show will be held at the White City Stadium, London, from Friday, July 22nd, to Friday, July 29th, inclusive. The evening of Saturday, July 23rd, and one other evening period during the Show will have to be given up to greyhound racing, but nevertheless these dates will result in a longer Show by four sessions.

The schedule will be based very largely on the classes held this year, but it may be possible to include additional classes. The first consideration of the extra time available will be to ensure that evening sessions will finish by 11 o'clock at the latest. Schedules are expected to be ready by March, 1949. There will be displays, but details of these have not yet been decided.

The note proceeds: "The 1948 Show was generally considered to have been a great success. There were 325 exhibitors as opposed to 264 in 1947, and the total entries were 1,087 as against 1,055. It is much regretted that the Show was not privileged by a visit of Their Maiesties owing to it being held in August, but an increase in the total gate of over 14,000 was indeed satisfactory. Jumping in the International Competitions was very keen and prize money on the whole was well divided. It was a great pleasure to have the opportunity of extending hospitality to six teams from abroad, and Colonel Thompson and the United States team are to be congratulated on winning the Prince of Wales trophy.

"Thanks are due to the National Pony Society for staging their successful and instructive parade of mountain and moorland ponies, and to the British Field Sports Society for their co-operation in making parades of foxhounds and beagles possible.

"Perhaps the highlight and most exciting moment was Colonel H. M. Llewellyn's great British victory in the King George V Gold Cup on his horse 'Foxhunter.'"

* * * * *

ALLEGATION OF CRUELTY TO CALVES

A report issued by Carlisle's Veterinary Inspector, Mr. W. G. Robinson, condemns alleged cruelty to young calves caused by exposing them in the grading stations at Carlisle, Longtown and Wigton, and transporting them alive by road to slaughter-houses as far away as Manchester.

"It seems to me," he states in the report, "that these calves probably go for at least 24 hours without food or drink. If it is necessary to slaughter them, would it not be possible to do this locally and transport the carcases?" he asks.

The Carlisle Health Committee have decided to bring the report to the notice of the Ministry of Food and other appropriate Ministries, with the strongest possible representations.—*Yorkshire Post.*

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

Correspondents are requested to write as briefly as possible.

NEW LABORATORIES OPENED AT ROWETT INSTITUTE

Sir,—In connection with your paragraph on the above in the issue of November 20th, it may interest members to know that there is one veterinary surgeon only on the staff of the Institute, and he is a specialist in ruminant physiology.

A strange state of affairs, surely, for an enterprise so largely devoted to work on farm animals and scarcely in keeping with the profession's supposedly new standing in that field.

Yours faithfully,

JOHN C. GRANT.

Veterinary Department,
Marischal College,
Aberdeen.
November 22nd, 1948.

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THE NATIONAL VETERINARY BENEVOLENT AND MUTUAL DEFENCE SOCIETY

Sir,—With reference to the case in which I was involved, which was reported in *The Veterinary Record* of November 20th, I should like to point out that the defence was undertaken by The National Veterinary Benevolent and Mutual Defence Society.

This defence was very ably carried out and it would possibly help the "Society" if you would kindly publish this letter of appreciation of their guidance and help throughout to my late partner and myself.

Yours faithfully,

A. SHENTON.

Trentburn,
St. Bernard's Road,
St. Albans, Herts.
November 27th, 1948.

DISEASES OF ANIMALS ACTS, 1894 TO 1937, AND AGRICULTURE ACT, 1937 (PART IV).

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Foot-and-mouth	Fowl Pest	Parasitic Mange*	Sheep Scab	Swine Fever
Nov. 1st to 15th, 1948	5	2	1	—	2	1
Corresponding period in—						
1947	7	—	397	—	—	2
1946	5	1	—	—	11	4
1945	7	4	—	—	5	22
Jan. 1st to Nov. 15, 1948	98	4	253	1	54	25
Corresponding period in—						
1947	104	103	1,797	1	74	33
1946	82	35	—	7	85	336
1945	110	123	—	6	108	867

NOTE.—The figures for the current year are approximate only.
* Excluding outbreaks in Army Horses.